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Human heredity...

843 ① human heredity
② human genetics
③ variations
④ inbreeding
⑤ racial differences
⑥ human races
⑦ human beings
⑧ " body
⑨ " health
⑩ " diseases
⑪ morbific heredity factors
⑭ psychopathy
⑮ psychological differences

HUMAN HEREDITY

by

ERWIN BAUR, EUGEN FISCHER,
AND
FRITZ LENZ

translated by

EDEN & CEDAR PAUL

with 172 illustrations in the text and 9 plates

TRAL FOOD TECHNOLOGICAL RESEARCH INSTITUTE, * MYSORE. *
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27 SEP 1950

LONDON: GEORGE ALLEN & UNWIN LTD
NEW YORK: THE MACMILLAN COMPANY

The German original of the present work is entitled "Menschliche Erblichkeitslehre," and the third edition was published by J. F. Lehmanns Verlag, Munich, 1927. The translation was made from that edition, but with supplements and corrections supplied by the authors during the preparation of the English version.

FIRST PUBLISHED IN ENGLISH IN 1931

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PRINTED IN GREAT BRITAIN BY
UNWIN BROTHERS LTD., WOKING

AUTHORS' PREFACE TO THE THIRD EDITION

THE science of human heredity has made great progress since the second edition of this book was published in the year 1923. To-day, far more trustworthy information can be given regarding the hereditary transmission of many diseases and other qualities, than was possible four years ago. An almost incredibly large number of new works has had to be taken into account. Since 1923, the non-German literature of the subject has become much more fully available than it was in war time and during the first few years after the war. For this reason, the references to such literature have been extensively amplified.

Comparatively little change has been requisite in Baur's section, dealing with the general theory of heredity, for in this department the elements of our science have been fairly well stabilised. But even in the more specialised field of human heredity, a like stabilisation is already in progress, so that we may hope that future editions of this book will not need so radical a revision.

In Fischer's section, additions have been made concerning the various forms of bodily structure or types of constitution, and also concerning the so-called blood-groups. In the illustrations, those of some of the Nordic types have been supplemented, or replaced by better ones.

In the sections contributed by Lenz, numerous genealogical trees have been added. It has been possible to formulate a general rule regarding the inheritance of morbid predispositions as contrasted with normal. The group of allergies or idiosyncrasies has been described in such a way as to show the inter-connexions between its members. Close attention has been paid to the hereditary determination of criminal tendencies. A good deal of new matter has been introduced into the chapter on the origination of hereditary morbid predispositions, as concerns the origin and frequency of these. In respect of methodology, guidance has been given for the calculation of regression and correlation. In view of the growing importance of the subject, special attention has been paid to the study of twins. The section on the inheritance of intellectual gifts has been considerably enriched. The relationships between racial characteristics and constitutional characteristics have been brought into association with the signs of degeneration and with those of exceptional aptitude. The chapter on the psychological differences between the leading races of mankind has been amplified by an account of the intelligence tests applied to soldiers in the United States army. A discussion of the relationships between race and criminality has also been introduced into this chapter.

ERWIN BAUR
EUGEN FISCHER
FRITZ LENZ

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INTRODUCTION

Every people or nation is subject to perpetual changes in its composition. The inhabitants of Rome at the close of the days of the empire were completely different from those who dwelt there in the time of the elder Cato—in respect not only of manners and customs, but also of general character. In like manner, the inhabitants of latter-day Germany are in their mental and bodily racial qualities totally different from the German contemporaries of Charlemagne. This development, this transformation of a people may be beneficial to it, may make it fitter, may signify a genuine advance; but it may (and most often actually does where civilised nations are concerned) denote a slow or speedy process of decay, of degeneration.

Those who are guided by the radically unsound analogy between a nation and an individual are apt to believe that every civilised people must pass, as it were, through phases of youth, maturity, and old age, being thus necessarily subject to degeneration, being thus foredoomed to perish. This is certainly not the case. But every nation is liable to morbid processes (selective, for the most part) which may rapidly modify its composition for the worse, and bring about its decay.

Most educated persons, nowadays, are familiar with the notion that such processes are a grave menace to our own as to other civilised races; that we, like the others, are in a degenerative phase. Attempts, injudicious as a rule, have already been made to devise and to apply social and legislative remedies for this "disease of the body politic".

Just as scientific therapeutics is impossible without a thorough grounding in human anatomy, physiology, and pathology, so there must be a broad foundation of natural science for the study of human sociology, for any fruitful attempt to solve the problems of population, and for our endeavours to promote racial hygiene (eugenics).

In all discussions concerning the effects of social and political influences upon the constitution of a nation, we shall be groping in the dark unless we know of what diverse racial constituents this nation is composed, unless we are acquainted with the laws governing the inheritance of racial distinctions and the innumerable differences between individuals, and unless we have discovered how selective processes influence a national stock. Legislative interference in matters of population and racial hygiene is, in default of such scientific knowledge, no more than the dangerous quacksalvery of uninstructed laymen.

The recognition of this fact has guided the arrangement of the contents of the present volume. The first section contains a survey of the general theory of variation and heredity. The second discusses the theory of variation and heredity in relation to the human species in particular, including the anthropological distinctions of race. The third section deals with the chief hereditary diseases and anomalies; the fourth, with the methods for the study of human heredity; and the fifth, with the inheritance of intellectual gifts. These five sections comprise a unity, in so far as they provide a theoretical demonstration of all the facts upon which individual human heredity depends. The present volume thus forms the scientific groundwork for a later treatise, *Human Selection and Racial Hygiene*, wherein will be considered the practical inferences from the theoretical knowledge now being acquired. In that book we shall discuss, first of all, the selective processes actually at work upon our populations; and we shall show that the hereditary composition of these populations is unquestionably being changed for the worse. The concluding sections of this subsequent work will deal more especially with applied racial hygiene (eugenics); will study how the process of racial degeneration is to be counteracted, and in what way the racial stock can best be improved. More particularly we shall show how the individual human being and the individual family can regulate their activities towards eugenic ends.

SECTION ONE

SKETCH OF THE GENERAL THEORY OF VARIATION AND HEREDITY

by

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CHAPTER ONE

FUNDAMENTAL NOTIONS

By inheritance in the biological sense of the word we denote that the offspring of the parent, or, in the case of sexual reproduction, of the parents, are like the parent or the parents as the case may be.

In multicellar living organisms, offspring may arise:

1. by the detachment and the independent further development of a single cell, or, in the case of organisms with multinuclear cells, of part of a cell;

2. by the detachment and independent further development of groups of cells or of entire organs;

3. by the detachment of two cells from one individual or from two individuals and the subsequent union of these two cells ("ovum" and "spermatozoid") to form a single cell, out of which a new individual proceeds.

In the first two instances, reproduction is said to be asexual or vegetative, and in the third instance it is termed sexual reproduction.

In many organisms, and especially in a great many vegetable organisms, any cell or group of cells can break away from the preexistent union. It thereupon takes on independent growth, so that a new individual[1] comes into being. In a great many organisms, however, only a few of the cells are endowed with this "capacity for universal regeneration". In yet other organisms, and above all in the higher animals, we find as a rule that only particular cells, the reproductive cells, are competent to generate new individuals.[2]

Manifestly in the organisms belonging to the first two groups, every one of these cells endowed with the capacity for universal regeneration, and in the organisms belonging to the third group every reproductive cell at least, must contain all the elements essential to the species concerned. In the intimate structure of the ova of an orang-utan and of a human being, respectively, there must somehow be present all upon which the differences between orang-utans and human beings depend. We must be careful, indeed, to refrain from picturing this to ourselves in the childish way characteristic of the preformation theory, according to which all the differential characteristics of the future adult organism were supposed to exist in miniature in the ovum. Every adult organism is the final outcome of a lengthy process of individual development; slight differences in the early stages may produce far-reaching differences in the later stages, the causes being small but the effects great. We can easily imagine that trifling differences in the make-up of two ova that are outwardly indistinguishable (chemical differences, perhaps) may make one of them develop into a European and the other into a Hottentot. But this does not necessarily mean that there is a precise relation between the extent of the differences between the germ cells and the extent of the differences between the adult organisms.

---

[1] Obviously in the case of such organisms the idea of individuality can hardly be said to be applicable in the strict sense of the term.

[2] In the higher animals, the only process comparable to the asexual reproduction of lower animals is the faculty possessed by embryos of dividing during the initial phases of their development, so that two or more individuals can arise out of one fertilised ovum. Thus twins, triplets, etc., are produced from a single ovum. This process, rare in human beings, is normal in many animals—armadillos, for instance.

Without any premature introduction of detailed cytological notions, let us use the term *idioplasm* introduced by Naegeli to denote that part of a cell upon which (in some manner which remains to be elucidated) its specific qualities depend. From this outlook, all the inheritable differences between two adult individuals are in the last analysis the outcome of structural or chemical differences in the idioplasm. These differences in the idioplasm are primary, whilst the obvious differences between adult organisms are altogether secondary. Any little difference between the idioplasms of two ova may, in the mechanical working-out of the developmental process. perhaps give rise to a whole series of differences in the most various parts of the adult organisms.

The transmission of hereditary characters from parent or parents (as the case may be) to offspring depends upon the fact that the latter have, wholly or partly, the same idioplasm as the parent, or in the case of sexual reproduction the parents.

CHAPTER TWO

THE PHENOMENA OF VARIATION

INHERITANCE is hardly ever complete: very rarely indeed can we say that the offspring are exactly like the parent or parents; and the members of a brood almost always differ one from another. As the phrase runs, the offspring "vary". The most important task of the science of heredity is to throw light on the laws of variation.

The causes of the differences between children and their parents, and of children in the same family one from another, can be classed for practical purposes under three heads, so that we distinguish three groups of variations:

1. Some variations depend on this, that individuals which qua idioplasm are completely identical, individuals which do not differ at all in respect of hereditary equipment, may vary because of their "peristasis", because of the varying circumstances in which they have developed. Variations of this kind are termed *modifications* or *paravariations*.

2. A second cause of variation is that in sexual reproduction the new individual is almost always engendered by the union of two cells whose idioplasms or hereditary equipments differ, so that we have a mingling, a combination, of two hereditary trends. Variations thus produced are termed *combinations* or *mixovariations*.

3. A third cause of variation is that, for one reason or another, and at one time or another, there occurs a change in the make-up of the idioplasm (which is as a rule fairly constant), leading to the formation of cells and consequently of individuals with a modified idioplasm—individuals which exhibit corresponding changes in their secondary external characters as well. Variations that are the outcome of such changes in the idioplasm are termed *mutations* or *idiovariations*.

1. MODIFICATION (PARAVARIATION)

To study the laws of modifications, we must begin with instances in which the causes of variation are simple and not complex, and above all with instances in which the effects of the sexual mingling of two hereditary trends have been excluded. When, therefore, we wish to deduce the chief laws of paravariation, we shall do well to choose an organism in which reproduction is permanently or at least temporarily carried on without the sexual union of two different idioplasms.

A group of organisms all of which have been derived from a single individual by vegetative reproduction is known as a *klon.* Let us take as example a klon of Paramaecium caudatum, a small unicellular infusorium commonly found in pools and puddles. This organism, when the water in which it flourishes is kept in a glass container, reproduces itself by fission generation after generation in a purely vegetative way. If, consequently, we people an aquarium with a single specimen to begin with, there will soon be a swarm of paramaecia all provided with the same hereditary equipment, all idioplastically identical. Yet in outward aspect the individuals of such a klon may differ markedly one from another, for they will have developed under different conditions. One, for instance, will always have secured enough food; another less fortunate will have gone hungry; one will have sustained an injury, another not; one will have inhabited a specially warm or a particularly well-lighted part of the container; and so on.

Definite laws apply to the differences among organisms with the same hereditary equipment, differences which are due to variations in the external influences that have been at work. If we select for statistical examination some particular quality common to all the specimens of such a swarm, we shall usually find that specimens diverging far from the mean in either direction are rare, but that the nearer we approach to the mean, the greater the number of individuals. This is shown by Table I, giving measurements of the length of the members of a klon of paramaecia. Fig. 1 shows the same statistical material set out in a curve.

TABLE I

Length in μ	136	140	144	148	152	156	160	164	168	172	176	180	184	188	192	196	200
Number of specimens	2	5	5	14	26	27	40	52	39	32	26	14	12	3	2	1	

It is easy to grasp why curves of variation should so often assume the form shown in the figure, a form corresponding more or less closely to that of the binomial curve. Confining our attention to the present instance, we know that the size of the paramaecia is influenced by all sorts of factors. For example, a paramaecium will not attain an exceptionally large size unless it is particularly well nourished, escapes mechanical injury, is kept in water containing a liberal supply of oxygen in solution, and is not exposed to too glaring a light or to

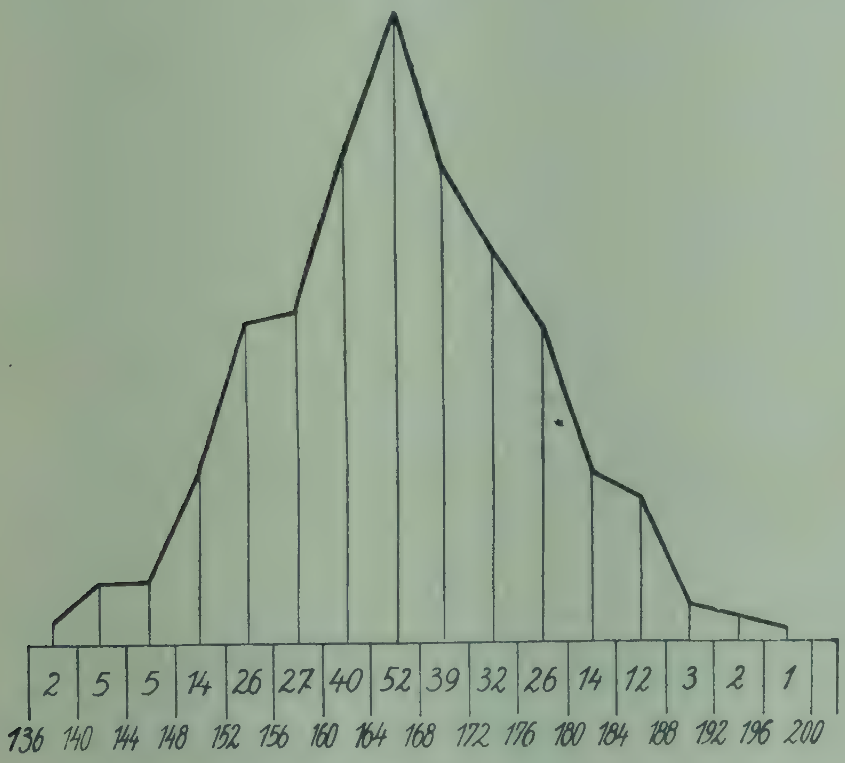


FIG. 1.

extremes of temperature or to other unfavourable conditions. Only a specimen that is fortunate in all these respects will grow to a very great length, and only one that has been unfortunate in all these respects will be seriously stunted. The creature's usual fate will be to have good luck in some matters and bad luck in others, so that its size will approximate to the average.

The question is, however, so important that it will be worth our while to consider in a more detailed way why the curve of variations assumes this special form.

The factors that may influence the size of a paramaecium in an aquarium are innumerable. Let us be content here to take into account the five already mentioned, and to assume that in the case of each of them there are only two alternatives, that of favouring or restricting growth in length. We shall denote the favouring alternatives by capital letters and the appropriate restricting alternatives by the corresponding small letters, as follows:

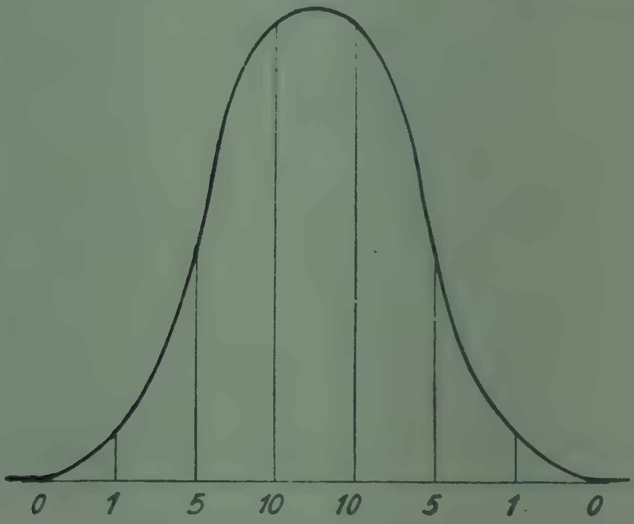


FIG. 2.

Favouring Alternatives	*Restricting Alternatives*
A abundance of nutriment	a scarcity of nutriment
B sufficient oxygen	b insufficient oxygen
C favourable illumination	c excessive glare
D no injury by cold	d transient injury by cold
E no injury by heat	e transient injury by heat

If we take into account no more than these five mutually independent factors, the individual specimens in a culture may be exposed to any one of 32 different assemblages of conditions. A specimen exposed during growth to the exclusively favourable conditions ABCDE will become exceptionally large, whilst one that is exposed to the partly favouring conditions AbcDe (being well nourished and not exposed to undue cold, but suffering from lack of oxygen, from excessive heat, and from glare) will not grow nearly so well. To simplify the problem, we will assume that each favouring factor is able to increase the creature's length by one unit, and we shall get the following results:

Possible Combinations of the five Mutually Independent Factors	Number of Units of Length added to the Adult Paramaecium under the Influence of this Combination	Possible Combinations of the five Mutually Independent Factors	Number of Units of Length added to the Adult Paramaecium under the Influence of this Combination
ABCDE	5	aBCDE	4
ABCDe	4	aBCDe	3
ABCdE	4	aBCdE	3
ABCde	3	aBCde	2
ABcDE	4	aBcDE	3
ABcDe	3	aBcDe	2
ABcdE	3	aBcdE	2
ABcde	2	aBcde	1
AbCDE	4	abCDE	3
AbCDe	3	abCDe	2
AbCdE	3	abCdE	2
AbCde	2	abCde	1
AbcDE	3	abcDE	2
AbcDe	2	abcDe	1
AbcdE	2	abcdE	1
Abcde	1	abcde	0

All of these 32 possible combinations are equally probable, and we may therefore expect that among a great number of specimens in an aquarium, 1 in every 32 will have developed under the influence of each of the combinations. But, as we see from the foregoing tabular statement:

there is 1 combination which produces an increase of length by 5 units
there are 5 combinations which produce ,, ,, 4 ,,
,, ,, 10 ,, ,, ,, ,, ,, 3 ,,
,, ,, 10 ,, ,, ,, ,, ,, 2 ,,
,, ,, 5 ,, ,, ,, ,, ,, 1 unit
there is 1 combination which produces ,, ,, 0 ,,

In other words we may expect to find that, among a large number of specimens,

$\frac{1}{32}$ will show an increase of length by 5 units
$\frac{5}{32}$,, ,, ,, ,, ,, 4 ,,
$\frac{10}{32}$,, ,, ,, ,, ,, 3 ,,
$\frac{10}{32}$,, ,, ,, ,, ,, 2 ,,
$\frac{5}{32}$,, ,, ,, ,, ,, 1 unit
$\frac{1}{32}$,, ,, ,, ,, ,, 0 ,,

Setting out this result in a curve, we get that shown in Fig. 2 on p. 28.

Had we taken into account 6 factors instead of 5, we should have obtained the numerical series 1, 6, 15, 20, 15, 6, 1; and any larger number of factors would in like manner furnish numerical series which, like

1
1 2 1
1 3 3 1
1 4 6 4 1
1 5 10 10 5 1

and so forth, would conform to the coefficient values $(a + b)_n$, thus corresponding to Gauss' curve of probability.

The characteristic form of the curve of modifications is therefore due simply to this, that there are numerous mutually independent factors of modification.

The curves of variation which are obtained when we undertake the statistical study of any quality are often more or less accurate reflexions of the foregoing mathematically calculated curves, but this is not always so. We may, for instance, get a curve with only one limb, a curve with several peaks, and so on. This happens because a change in one quality does not necessarily run parallel with changes in other conditions. But to follow up these questions would take us too far afield.

As far as plants are concerned, there has been plenty of experimental work to show in what way great differences between individuals with the same hereditary equipment may arise, and how extensive a modification may be; but as regards animals, and especially as regards higher animals, we have very little trustworthy material of this kind. To secure thoroughly uniform material in the case of the higher animals is extremely difficult; for in them, since reproduction is sexual, the offspring are always the fruit of two individuals. An instructive experiment of this kind in the case of one of the higher animals is shown in Fig. 3 (opp. p. 33).

When an organism is modified by the influence of the environment (by the influence of its "peristasis"), its idioplasm (its hereditary equipment) is in general unaffected. This can best be shown by referring once more to our example of paramaecium.

If from the paramaecia in an aquarium we detach a klon, we find that the individual specimens vary in size, but that, nevertheless, the smallest specimens and the largest ones are idioplasmatically identical, inasmuch as their offspring resemble one another in consisting of individuals of varying sizes. (See Fig. 4.)

We may conduct such a selection experiment to show variations in size for a number of generations, and we shall always get the same result; we shall always find that the special characteristics of the *individual* organism as regards size are not transmitted by inheritance, but that *each organism in the kinship inherits the entire characteristic modifiability of its kinship.*

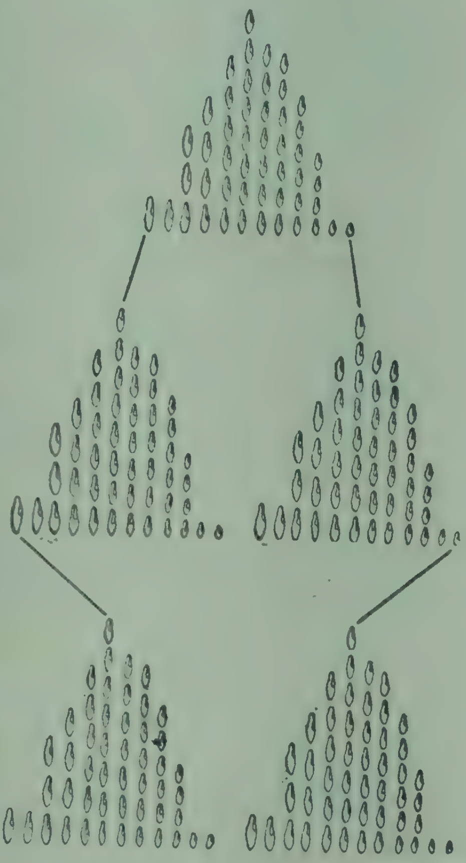


FIG. 4.

Diagram showing the result of a selection experiment with a klon of paramaecia. The offspring of the largest and of the smallest organism in the primary culture show identical variations in size. In each culture, organisms of identical size are ranged in vertical series, so that the frequency with which the various sizes appear can be easily made out.

Carrying out the experiment in a slightly different way, we can take two organisms from a klon and transfer one of them to an aquarium containing ample nutriment and the other to an aquarium containing an insufficient supply of nutriment. The result will be a great difference between the cultures in the respective aquaria. The individuals in the well-nourished culture will on the average be considerably larger than the individuals in the poorly nourished culture. The two cultures will differ greatly, although their hereditary equipments are identical. But if, after several generations of paramaecia have been bred in the respective aquaria, we then transfer to one and the same aquarium an individual organism taken from among the "fat kine" and another taken from among the "lean kine", we shall find that the offspring of these two, when placed in identical conditions, are themselves identical in respect of average size. Thus culture under diversified conditions has not affected the idioplasm of the klon, although the two strains seemed so different during several generations of culture.

A number of different kinships of Paramaecium caudatum can be fished out of every puddle, each of them endowed with its own specific modifiability, representing a definite *idiotypus*, which breeds true.

We find a like conformity to law in the case of sexual reproduction. This, however, is plainly recognisable only when we work with organisms which reproduce themselves "autogamically"; that is to say in many plants, such as beans (Phaseolus vulgaris), oats (Avena sativa), etc. In such plants the flowers are androgynous, and as a rule fertilisation is effected exclusively by pollen from its own flower reaching the stigma. A stock of plants originating through self-fertilisation in normally autogamous plants, is known as a *pure line*.

Within a pure line, the mode of inheritance is identical with that which occurs within a klon (see above page 26). Here, likewise, modifications are not transmissible by inheritance, as can be plainly shown by experiments on the higher plants. In the Chinese primula, that well-known ornamental plant, there are races of various colours, and among them a red one and a white one, distinguished as Primula sinensis rubra and Primula sinensis alba, respectively. The colour of flowers, like other qualities, is subject to marked paravariations in consequence of all kinds of external influences, and especially owing to changes in temperature. If we put plants belonging to a simple "red" strain in a warm and rather shady conservatory kept at an average temperature of

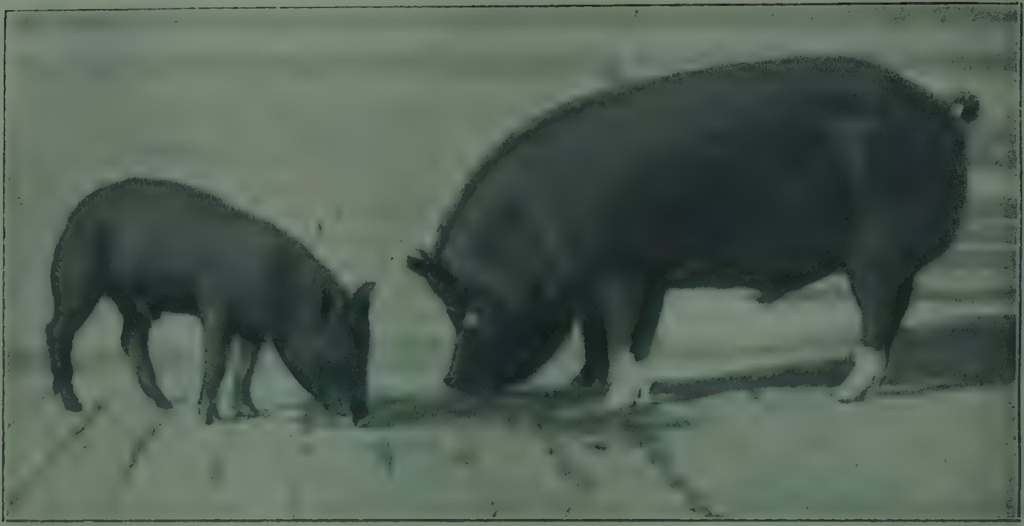


FIG. 3.—Two pigs from the same litter of an extremely uniform breed (Berkshire). The pig in the left of the figure is very poorly nourished, but the pig in the right of the figure is very well nourished. These are not inheritable modifications. (After Mathusius.)

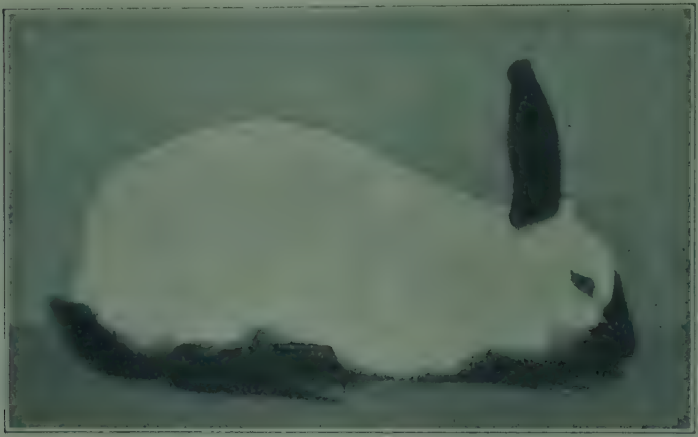


FIG. 5.—Himalayan rabbit

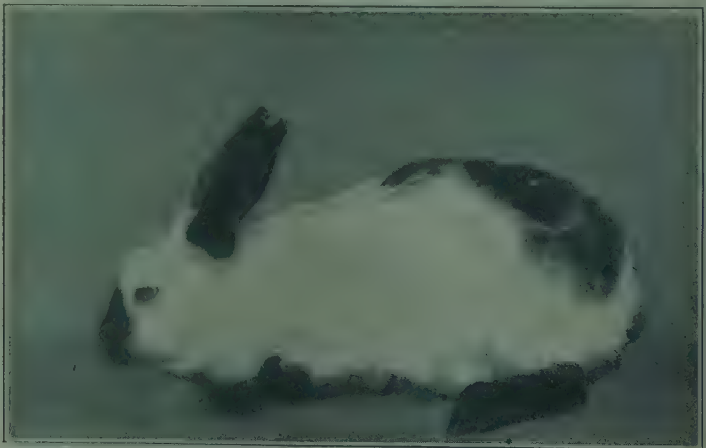


FIG. 6.—Himalayan rabbit in which an area of the back was chilled by shaving it, as a result of which all the hair newly grown on the shaven spot has a dark colour.

about 96° F., the flowers will be white, whereas the plants of the same strain kept at a temperature ranging from 50° to 60° F., will have red flowers. The offspring of such plants, which when cultivated in a hothouse are indistinguishable from those of a Primula sinensis alba, bear red flowers when cultivated once more under normal conditions, that is to say at a temperature ranging from 50° to 60° F. The inheritable idioplasmatic difference between the two races, therefore, is not that one bears "white" and the other "red" flowers, but that the two strains react differently to changes in temperature.

Paraffinum durum and paraffinum liquidum are for the layman distinguishable by this, that the former at ordinary temperatures is a solid white substance, whereas the latter, under the same conditions, is an oily fluid. But when the two paraffins are kept at a temperature of 140° F., they are just as little to be distinguished from one another by external characteristics as are the two strains of primula in a hothouse. The difference between the two paraffins is that they have different melting-points, this meaning that they react differently to the influence of temperature. Now, just as little as a "paraffinum durum" which is heated to a temperature of 140° F., thereby acquires a lower melting-point, thus becoming "paraffinum liquidum", just so little does a Primula sinensis rubra which bears white flowers because it is grown in a hothouse, become a Primula sinensis alba.

Let me insist once more that a hereditary distinction such as is characteristic of a particular stock is not a specific development of a character, but nothing more than a specific "way of reacting". Thus the aspect of an organism always depends upon two things: first, upon its hereditary ways of reacting, or, to put the matter in other words, upon its idioplasmatic constitution; and, secondly, upon its peristasis, that is to say upon the external influences to which it has been exposed during the course of its individual development.

It is strange how widely prevalent are confused notions concerning this matter.

Perfectly analogous experiments can be made in the case of higher animals. Among rabbits, for instance, there are two somewhat similar races, one of which is pure white with pink eyes, while the other, (the "Himalayan" rabbit) though mainly white and with pink eyes, has very dark fur on the ears, paws, tail, and nose. (See Fig. 5, opposite p. 33.) The colour of the fur in this latter race is modifiable by temperature. If an area of its skin be kept cool, which can easily be effected simply by shaving a part, all the new hairs that grow upon the cooled area have a dark tint. Fig. 6 (opposite p. 33) shows how a patch which had been thus shaved has been covered with dark hair. But as soon as the fur has regrown, so that the area of skin is now protected by it from the cold, all the new hairs which subsequently grow in the area are white, as before, so that the dark tint of the shaved area gradually disappears. It would be easy enough, by shaving the whole surface of the body, to provide one of these rabbits temporarily with a dark-tinted fur throughout; and, conversely, it is possible to make rabbits of this stock white all over, including the ears, the paws, the tail, and the muzzle, if they are kept at a sufficiently high temperature.

The idea that a change, a modification, in some visible characteristic of an organism, such as the colour of a primula, implies of itself a change in the hereditary modes of reaction of the stock, is one which, as the outcome of falsely interpreted observations, continues to haunt a good many minds, and finds expression in the catchword concerning the "inheritance of acquired characters".

The only thing that can ensure the avoidance of such a confusion is perfect clarity in the use of terms. An inheritable quality signifies nothing more than the "inheritance of a specific way of reacting" to external influences. Consequently, the origination of a new quality depends upon a modification in the previous way of reacting. If we heat a paraffinum durum not merely to 140° F., which will melt it, but to a much higher temperature, while simultaneously exposing it to considerable pressure, it undergoes a change in its chemical constitution, and may then, in certain circumstances, be transformed into a paraffin with a lower melting-point. In like manner, certain unusual influences, such as that of exposure to radium, or persistent cultivation at a high temperature which has no lasting effect when the plants are exposed to it only for a short time, may enable us to obtain from a Primula sinensis rubra an offspring which, as regards the colour of its flowers, will react differently to temperature from the original stock.

But just as the liquefaction of a paraffinum durum brought about by a change in chemical constitution and by a reduction of the melting-point is something altogether different from a simple melting, so, likewise, does the production of such a new race of primula signify something altogether different from a mere modification in the colour of the flowers due to cultivation in a hothouse.

A change of this latter kind is known as a mutation.

Unfortunately, the expression "the inheritance of a new quality" is still applied by many biologists to these two utterly distinct cases.

If, then, we are to take up a position as regards the thorny problem of the inheritance of acquired characters, we shall say:

Newly originating characters or qualities, even when their origination is brought about by external influences, are or may be inheritable when they are genuinely new characters or qualities in the before-mentioned sense; but merely because an organism undergoes a modification, it does not follow without further ado that this modification is also a mutation. The notion so firmly fixed in many peoples' minds that a modification invariably, or at least in most instances, involves an identical mutation, is the outcome of erroneously organised experiments and fallacious inferences.

The most frequent source of fallacy is to be found in the use of experimental material which is not hereditarily unifiable. The mistake is so easily made and is so common that it will be well to illustrate it by an example. As already said, in Paramaecium caudatum there are always numerous stocks, each of which is hereditarily constant. Fig. 7 shows a number of such paramaecium kinships. Every one of these eight kinships conforms to the laws of inheritance illustrated in Fig. 4. What each individual inherits is not its particular size, but its stock character. As a glance at Fig. 7 shows, the curves of modification of the different stocks overlap one another. If, therefore, we have these different stocks of paramaecium in the same aquarium, there is a continuous gradation in size from the smallest to the largest, and there is no obvious indication of the fact that there are eight hereditarily different stocks or klons in the aquarium. Even if the observer did know this, it would be impossible for him to tell by simply examining an organism of mean size to which klon it belonged. Organisms of this size are to be found in all the eight klons. Because two organisms are "paratypically" identical, it does not follow that they are likewise "idiotypically" identical. If we make a selection experiment with such mixed strains as are shown in Fig. 4, if from an aquarium containing such mixed strains we separate the smallest organism and the largest, then we shall certainly find that, on the average, the largest organism has larger offspring than the smallest organism has. For, as we see when we study Fig. 7, the largest organism certainly belongs to the uppermost of the kinships shown in the figure, and will transmit, though not its individual size, its stock character, so that it will have offspring which on the average are larger than the average size of the whole mixture. In like manner, the smallest organism will belong to the stock shown lowest in the figure, and will therefore have offspring of an average smaller size. But the fact that the selection of organisms differing widely from the general average results in a shifting of the mean size in the direction of that of the selected specimen, is no proof that the modification is inheritable, seeing that

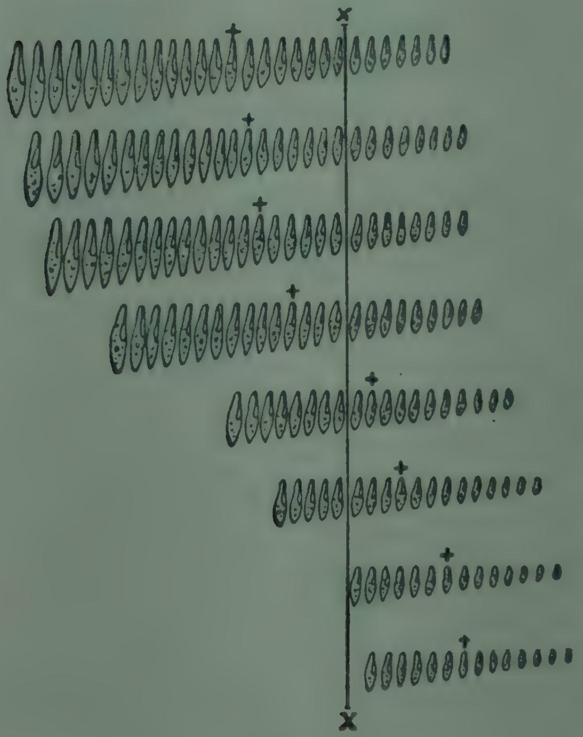


Fig. 7.

Eight distinct klons of paramaecium. The line X–X indicates the mean of the size of the organisms of all the eight klons. In each klon, the organism of mean size has a + mark above it. (After Jennings.)

the result of our experiment is solely due to the fact that the selection material was not unifiable in a hereditary sense. The organisms diverging strongly from the average do not transmit by inheritance an acquired modification, but merely transmit the stock character they already possess. In the literature concerning the inheritance of acquired characters, this fallacy plays a momentous part.

A second source of fallacy in this discussion is that very frequently external influences which have acted on an individual have also influenced its offspring more or less directly.

Such persistent influences can very often be observed. If we grow a great number of plants belonging to a pure line of beans, we shall find that the individual plants vary in size and strength according as the nutritive and other conditions vary. What is transmitted by inheritance in this case likewise is nothing but the stock character, so that the selection of larger or smaller plants within such a pure line does not lead to any change in the average size of the individual plants.

What applies to the paramaecia shown in Fig. 4, applies also to this experiment with beans. A bean plant can be half starved and can be supplied with an insufficiency of water, with the result that the beans which it forms are wrinkled and very small. From these beans there will then develop plants which, in the early stages, when they are living only on the supplies of nutriment derived from the mother plant, are very badly nourished, and they will therefore grow into manifestly weaker bean plants than the offspring derived under otherwise identical conditions from a plant of the same stock which has not been ill-treated. Obviously, in this case too, there has been no "inheritance of the individual modification" of the maternal plant. All that has happened is that the unfovourable nutritive conditions which have already influenced the mother, continue to act upon the children in the earliest stage of their development. A generation later (if nutritive conditions are favourable) we shall find that the after-effects have ceased, and that the beans are no longer influenced.

In higher animals, the persistent effects of such external influences are far more striking. We shall easily understand this when we bear in mind that in all viviparous animals the embryos pass through the most important part of their development within the maternal body. Furthermore, in many animals, a sort of "anticipatory" development takes place even in the ova, a number of developmental processes being initiated here, and definite "organ-forming substances" and "organ-forming regions" appearing in their protoplasm. In these cases, too, therefore, a large part of the development, even in oviparous animals, goes on within the maternal body, being thus subjected to the influence of the external conditions which act on the mother.

This sort of fallacy has played a great part in the much discussed experiments of Kammerer, not a single one of which can be uncritically accepted.

When these and all other sources of fallacy have been excluded, the invariable result of experiment concerning the inheritance of modifications has been to show that no such inheritance occurs.

It is, of course, possible that one and the same cause may sometimes chance to bring about simultaneously a definite modification and an identical mutation. For instance, it might happen that the cultivation of a Primula sinensis rubra in a very hot conservatory would result in a mutation of the idioplasm as well as in a modification of the flowers. It is also possible that, by chance, the result of this mutation would be the production of a race which would bear white flowers even at lower temperatures. But not a solitary instance of such a coincidence has ever been recorded, and mathematical calculation will show that the likelihood of any such coincidence is exceedingly small. Certainly there can be no question that such a coincidence between modifications and mutations must be infrequent, and cannot possibly occur as a general rule!

Grafting experiments demonstrate very clearly that the idioplasm of the reproductive cells is beyond the reach of influences acting on the body which carries them. If, for example, we graft gillyflowers upon a Brussel-sprouts stem, or a white rose upon the stem of a red rose, the offspring of these grafted plants breed true to the original race.[1] The same thing applies to animals. The ovaries can be removed from a young caterpillar of Ocneria dispar and transplanted into a caterpillar of another race of Ocneria. They will heal in, and subsequently, in the sexually mature imago, will produce ova belonging to the pure race of the caterpillar from which they were taken. The body of the animal of the other race, in which they have been carried for months, and in which they have first grown to maturity, has exercised no influence whatever upon them or upon the animal developing out of them. Similar experiments have been made in mammals. For example, we can remove the ovaries of a white rat and replace them by the ovaries of a black rat.[2] This white rat will henceforward produce young belonging to the black-rat stock.

The foregoing laws apply to all organisms hitherto studied, the higher animals not excepted. We have, therefore, every reason to believe that they apply no less to the human species. As yet, however, no trustworthy observations have been carried out in the case of man.

Nevertheless, everyday observation shows that the bodily and mental qualities of an adult human being are the outcome of two things: first, his hereditary equipment, that is to say his idioplasmatic constitution as transmitted by inheritance; and, secondly, the external influences to which he has been exposed in the course of his individual development—nutritive influences, educational influences, and the like. It is extremely probable that climatic influences (in the widest sense of the term) induce modifications in human beings. This no doubt partly accounts for the fact that the persons belonging to one nation—comprising a medley of races living together in a circumscribed area—often exhibit a marked similarity in their bodily and mental qualities, however difficult it may be to describe this similarity in precise terms.

[1] Isolated flower-buds, and even part of a flower, can be grafted upon individuals belonging to another race or another species and will grow to maturity.

[2] The ovary is implanted into the cavity of the uterus shortly after a delivery. In most cases it heals in perfectly, and the foreign ovary functions satisfactorily in its new surroundings.

In our study of human heredity we must never fail to bear in mind that what an individual transmits by inheritance is not the qualities he has himself personally developed during his lifetime, but only the hereditary constitution which he himself received from his parents. For example, a rupture is not transmitted by inheritance, but only "a certain predisposition to become ruptured". Whether one who inherits this predisposition to rupture will actually become ruptured, depends upon other things than inheritance. But as regards the further transmission of the predisposition by inheritance it is a matter of no moment whether one who has inherited such a tendency himself becomes ruptured owing to excessive muscular exertion or for any other reason. He who remains objectively sound will nevertheless transmit the predisposition to his offspring. Where we are concerned with a hereditary predisposition to a disease whose onset during the lifetime of the individual is determined by a rather infrequent concatenation of external influences, it is, as the foregoing considerations will show, very difficult to trace the course of inheritance precisely. Let us suppose, for example, that in a family there is a moderate predisposition to rupture. Then only a few members of the family, only a few of those who have the hereditary predisposition, will actually develop a hernia—only those who have made exceptional exertions or have in some other way been especially subjected to the exciting causes of rupture. One who studies the family history in such cases will be apt to think that the hereditary transmission of the trouble has been irregular and "capricious", whereas in reality the transmission has been perfectly regular—for what has been transmitted is not the individual occurrence of rupture, but only the predisposition thereto.

We must also bear in mind that every human being goes through a very large part of his development within the mother's womb, and that environmental influences acting on the mother may also be weighty with consequences for the unborn offspring. Here we have to do with "after-effects", such as we have already discussed above on page 37.

Let us suppose that a woman who is not herself unduly predisposed to tuberculosis, one whose power of resisting tubercular infection is not below average, has nevertheless become tubercular owing to the unfavourable nature of her occupation; and let us further suppose that owing to her illness her personal nutritive conditions are about as bad as they can be. Now, the child of such a woman will in many cases be weakly from birth upwards, will have a power of resistance that is below average, and will therefore be exceptionally apt to became infected with tubercle. Here hereditary transmission will be simulated, when in reality we are concerned exclusively with after-effects. In an outwardly similar case, when a woman with a hereditary predisposition to tuberculosis—determined by a peculiar shape of thorax, or by some deficiency in the lymphatic system, or in some other way—becomes tubercular and procreates tubercular offspring, heredity will play a part in addition to after-effects. To ascertain the real determinants in such cases is very difficult, and often impossible. In all investigations of the kind, we must draw our inferences with the utmost caution, and must subject our data to the shrewdest criticisms.

No more in human beings than in any other organisms are modifications transmissible by inheritance. Nevertheless, the most extraordinary notions concerning this matter are widely diffused. By careful training, a child without hereditary musical endowment may be equipped with a fair understanding of music, and may even become a creditable musical performer; but there is absolutely no warrant for the belief so often expressed in popular writings on the subject that the offspring of such a musically-trained person will start in life with a better musical endowment than their parent. There is no warrant for the belief that hereditary endowments can be increased merely by education. *Education has very remarkable effects upon the individual during that individual's lifetime, but it does not influence the hereditary endowment of the offspring.* If a nation or a particular stratum in a nation has an inferior hereditary equipment (the Negroes, for instance, in the United States of America), education and cultural influences may improve individual members of the nation or the stratum, but they cannot alter the stock.

The recognition that the extant hereditary equipment of an individual must be regarded as almost inalterable, and the recognition that between members and races of the human species just as between members and races of lower animals and of plants there exist considerable hereditary differences which cannot be modified or eliminated by rearing and education—conflict sharply with preconceived opinions which are widely current. But neither the biologist nor the physician must allow his scientific insight to be clouded by such political prejudices!

It need hardly be said that attempts will be made to influence the extant constitution of a people in both ways: on the one hand by the best possible rearing, education, and applied hygiene, that is to say by all the methods of "individual hygiene"; and, on the other hand, by methods aiming at the improvement of the racial stock, that is to say by the methods the authors propose to discuss in their subsequent work on human selection, eugenics, or "racial hygiene". In like manner, the agriculturist tries to secure the best possible results in two different ways; first, by the use of the best manures and by caring for his plants as well as he possibly can; and, secondly, by the purposive increase of the functional capacity of these plants—that is to say by breeding.

2. COMBINATION (MIXOVARIATION)

a. General Considerations

In sexual reproduction, the hereditary constitutions of the two parents will as a rule be more or less different. Exclusively or almost exclusively in cases of self-fertilisation can the reproductive cells which coalesce in sexual reproduction have a perfectly identical hereditary equipment—only, that is to say, in those hermaphrodite plants and animals in which a male and a female reproductive cell from the same individual coalesce. But when the two conjugating reproductive cells have different hereditary equipments, the course of the process of inheritance is far more complicated, and these complications are the source of the phenomena of variation which are known as combinations (or mixovariations).

That the reader may become acquainted with the laws which govern such instances, let us begin by the consideration of a simple case. Let us cross two specimens of Antirrhinum majus (the garden snapdragon), one of them being a plant of pure race bearing ivory-coloured flowers (a, Fig. 8) and the other being a plant of pure race bearing red flowers (b, Fig. 8). We will use certain letters to distinguish the two plants. Each plant arises out of the union of two reproductive cells. Let us call a reproductive cell belonging to the red-flowering stock *F*, and a red-flowering plant which originates out of the union of two such cells *FF*. In like manner, we will call a reproductive cell belonging to a plant of the ivory-flowering stock *f* and a plant of that stock *ff*. When we produce a hybrid between a red plant *FF* and an ivory plant *ff* by effecting a union between a female reproductive cell *F* and a male reproductive cell *f* or (for this is indifferent) a female reproductive cell *f* and a male reproductive cell *F*, we get a plant which must be designated *Ff* or *fF*—that is to say, a hybrid, or, to use the technical term, a "heterozygotic" individual, one produced by the conjugation of reproductive cells differing in hereditary equipment. In contrast with this, we term an organism which has originated out of the conjugation of two reproductive cells having like hereditary equipments, "homozygotic". Now, such an Antirrhinum hybrid will neither be ivory-coloured, like one of its parents, nor red like the other, but will bear pink flowers (c, Fig. 8). It has inherited from only one of the parents, the red one, the "capacity for forming the colour of red flowers", with the result that its flowers are of a much paler red than those of the red parent, being pink. This, perhaps, is what might have been expected; but if so the behaviour of the offspring of such a hybrid will seem all the more striking. When we allow a number of such hybrids to fertilise one another, or when we allow them (as can be done without detriment) to fertilise themselves with their own pollen, we get plants of three different kinds. Some of these hybrid offspring have red flowers, of exactly the same tint as those of the original red parent stock; some of them have ivory-coloured flowers, just like those of the original ivory parent stock; and some of them have pink flowers, like those of the hybrid generation from which they have sprung. When we experiment with sufficiently large numbers of plants, we

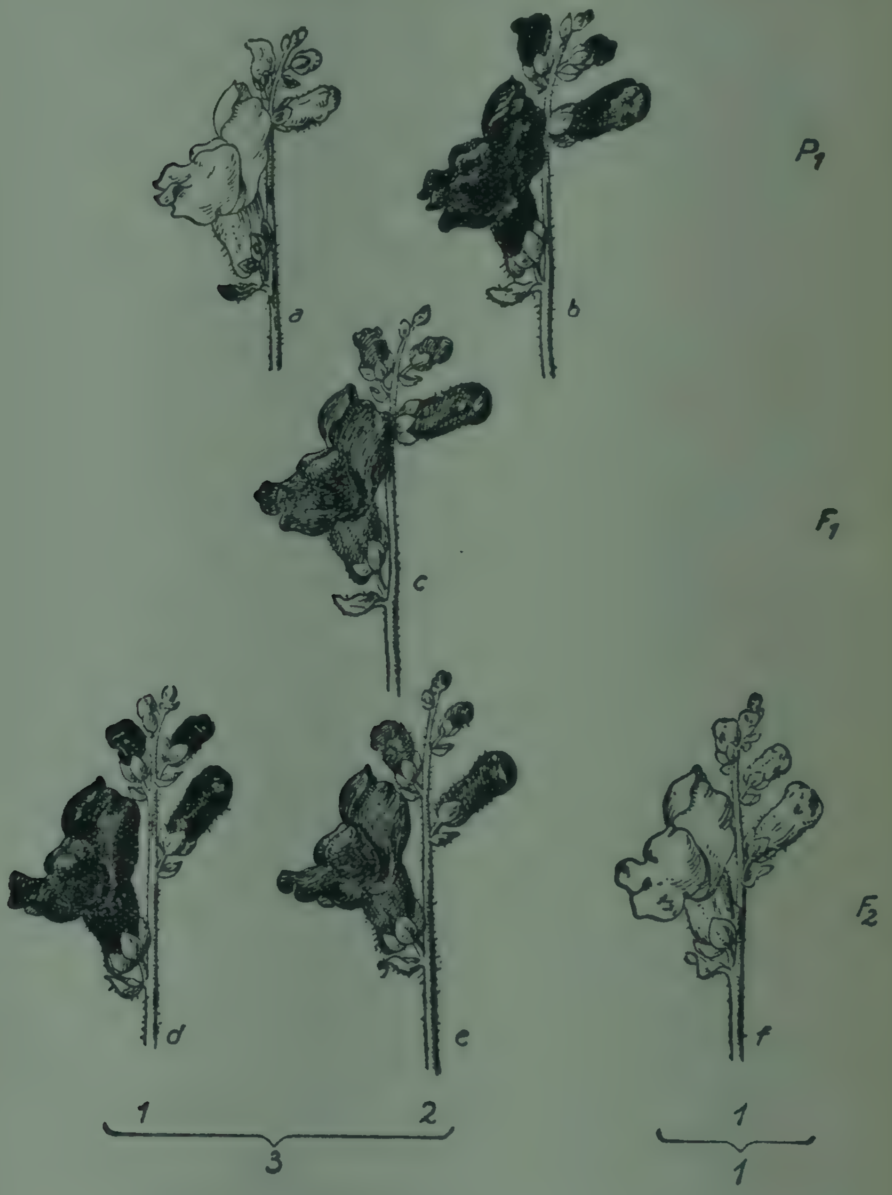
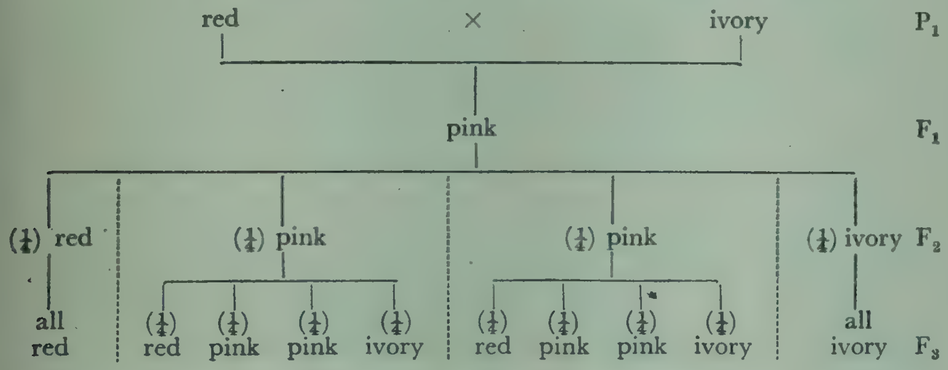


FIG. 8.

The crossing of an ivory-flowering stock with a red-flowering stock of the garden snapdragon (Antirrhinum majus). The hybrid (c) is pink; the offspring of the hybrid, obtained by self-fertilisation or by mutual fertilisation on the part of two such hybrids, consists of red-flowering (d), pink-flowering (e), and ivory-flowering (f) plants, in the proportion of 1 red : 2 pink : 1 ivory.

find that those of the second generation from the original cross are always grouped numerically in the proportions of one red to two pink and one ivory plant. We find, that is to say, among a hundred plants of the second generation, about 25 reds, 50 pinks, and 25 ivories. The actual figures in one particular series of experiments were that among 97 plants, 22 were red, 52 were pink, and 23 were ivory. Carrying the experiment a generation farther, we find that the red plants of the second generation have exclusively red offspring, that the ivory plants of the second generation have exclusively ivory offspring, but that the pink individuals of the second generation behave exactly like the pink specimen of the first hybrid generation *Ff*, that is to say that they once more produce red, pink, and ivory offspring in the proportion of 1 : 2 : 1. Setting this out as a genealogical tree, we get the following result:



The capital letters printed at the right-hand side of the foregoing table, like those at the right-hand side of Fig. 8, have the following significance:

P_1 is the first parental generation, that is to say it consists of the individual originally used for the crossing; F_1 is the first filial generation, that is to say it consists of the hybrid of the primary generation; F_2, F_3, etc., are the subsequent hybrid generations. In like manner, P_2 will be used to denote the earlier parental generation from which P_1 was derived, and so on.

Mendel's theory, now generally accepted, enables us to understand these peculiar results of crossing. According to that theory, every such hybrid forms two kinds of reproductive cells, 50 % of them being "paternal" and 50 % "maternal". Thus, on this theory, our Antirrhinum hybrid *Ff* forms two kinds of ovules and pollen grains, half of which are wholly of the kind *F* and thus behave in exactly the same way as the reproductive cells of a homozygotic *FF* plant, while the other half are precisely identical with the reproductive cells *f* of the ivory-flowering stock *ff*. When we fertilise the hybrid with its own pollen, or when a number of such hybrids fertilise one another, the two different kinds of reproductive cells, *F* and *f* respectively, can unite in four different ways:

an ovule *F* may encounter a pollen grain F producing a plant *FF* which will bear red flowers;
an ovule *F* may encounter a pollen grain *f* producing a plant *Ff*, which will bear pink flowers;
an ovule *f* may encounter a pollen grain *F* producing a plant *fF*, which will bear pink flowers;
an ovule *f* may encounter a pollen grain *f*, producing a plant *ff* which will bear ivory flowers.

All four of these possible encounters are equally probable, and we may therefore expect that among the offspring of such a hybrid all four possibilities will be realised with equal frequency, with the result that the offspring will consist of four varieties of plants, *FF*, *Ff*, *fF*, *ff*, which will be formed in practically equal proportions. Theoretically, therefore, we may anticipate that the offspring of such a hybrid will be composed as follows:

¼ of plants originating as $F \times F$ and therefore homozygotically red;
,, ,, ,, originating as $F \times f$ and therefore heterozygotically pink;
,, ,, ,, originating as $f \times F$ and therefore heterozygotically pink;
,, ,, ,, originating as $f \times f$ and therefore homozygotically ivory.

On the basis of this theory it is possible to predict the result of further experiments, and thus to test the soundness of the theory. This especially applies to the recrossing of a hybrid. Let us fertilise specimens of our hybrid *Ff* with pollen from one of the ivory parental plants *ff* or from another ivory plant of the same stock. According to the hypothesis we are considering, the hybrids form two kinds of ovules, half of the ovules transmitting exclusively the characteristic of producing red flowers, and the other half transmitting exclusively the characteristic of producing ivory flowers. When we have such a hybrid fertilised by a *ff* plant, whose pollen grain transmits exclusively the characteristic of producing ivory flowers, 50 % of the resulting offspring must be produced by the union of an *F* ovum with an *f* pollen grain. Consequently, as the outcome of such a recrossing, there must arise: 50 % of plants with the formula *Ff*, that is to say pink heterozygotes; and 50 % of plants having the formula *ff*, that is to say ivory plants, which will henceforward breed true. Now, the results foreshadowed by the theory do actually occur; as the outcome of an experiment of the kind, we obtain almost equal numbers of ivory-flowering plants that breed true, and pink-flowering plants that continue to show mendelian inheritance.

In the instance of hybridisation we are now considering, the hybrids can easily be recognised by the pink colour of their flowers. They occupy an intermediate position between the parents, and may therefore be spoken of as "intermediate" hybrids. Very often hybridisation takes this intermediate form, but by no means always. The aspect of the heterozygotes may be quite different from that of something intermediate between the parents.

Very frequently we find that the heterozygotes closely resemble one of the parents, are "goneoclin". This happens, for instance, when we cross a white mouse with a black one. Most of the resulting hybrids are black, and are not distinguishable in outward aspect from the homozygotic black parent stock. As far as outward aspect is concerned, F_2 of such a crossing consists of individuals $\frac{3}{4}$ of which are black and $\frac{1}{4}$ white individuals. But experiment shows that, among these seemingly black hybrids, some of them are homozygotically black, whilst others, $\frac{2}{3}$, are heterozygotically black. This is discovered by recrossing each of the black mice of F_2 with a white mouse. In these circumstances, $\frac{2}{3}$ of the black F_2 mice produce black offspring and white offspring in a ratio of 1 : 1, whereas the remaining third produces only black offspring. In reality, then, and outward appearance notwithstanding, like conditions obtain in the case of these hybrids

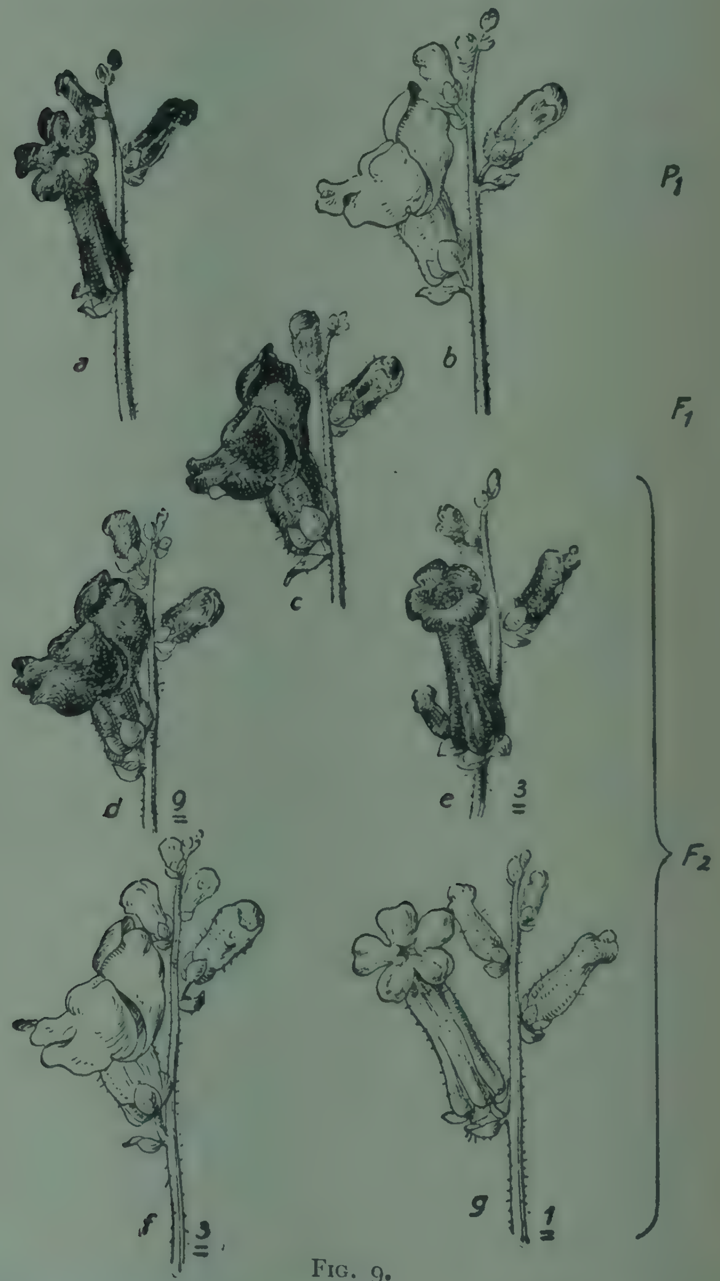


FIG. 9.

Crossing of a red peloric (a) with an ivory normal stock (b) of Antirrhinum majus. The hybrid (c) is pink normal. The F_1 generation consists of red and pink normal (d), red and pink peloric (e), ivory normal (f), and ivory peloric (g) plants, in ratios of 9 red normals: 3 red pelorics: 3 ivory normals: 1 ivory peloric. Red and pink flowers are not distinguished in this figure.

as in the case of the hybrids formed by crossing the red-flowering and the ivory-flowering snapdragon.

To denote the fact that many hybrids are in appearance indistinguishable from one of the parents, we use the term "dominance". We say that one character (in this instance, the "capacity for forming black hair-pigment") is "dominant" over another character, over the lack of this capacity—and we say that this latter characteristic is "recessive" as compared with the former.

Undue importance has been attached to this phenomenon of dominance, and some biologists have spoken of a "rule of dominance". This is altogether wrong. Dominance is by no means a universal phenomenon, and no one has any right to speak of a generally valid rule of dominance. Even when dominance is ostensibly complete, closer examination will often enable us to distinguish homozygotes from heterozygotes.

Sometimes, moreover, a dominant quality becomes more conspicuous or less conspicuous as the age of the hybrid increases.

Identical laws apply when we cross races which differ from one another in more than one character, so that the crossing leads to the production, not of mere "monohybrids", but of "dihybrids", "trihybrids", and "polyhybrids". Let us again choose a very simple case for consideration. We cross a red peloric[1] snapdragon stock (a, in Fig. 9) with an ivory snapdragon stock bearing normal snapdragon flowers (b, in Fig. 9). The hybrids will be pink and will have flowers of normal snapdragon shape, except that the upper lip will be a little smaller than in the homozygotic normal plant. Here, then, we have an instance in which the normal (not radially symmetrical) flower shape is completely dominant over the peloric, whereas in respect of colour the hybrids are intermediate between the parents.[2] The F_2 generation obtained from such

[1] When in plants which normally bear flowers that have lost the primitive radial symmetry of the typical flower, the flowers revert to the ancestral radial symmetry, such flowers, being "monstrous" for that particular species or genus, are said to be "peloric".

[2] In Fig. 9, the pink individuals and the red individuals are similarly shaded.

hybrids by self-fertilisation (d to g, in Fig. 9) consist of six outwardly distinguishable groups of plants, as follows:

red normals . . .	(3)	pink pelorics . . .	(2)
pink normals . . .	(6)	ivory normals . . .	(3)
red pelorics . . .	(1)	ivory pelorics . . .	(1)

The numerical ratios in which the plants of these various categories appear are shown by the figures in parenthesis. How these categories and these numerical ratios come about can be deduced in the same way as in the foregoing example. A reproductive cell or (to use the term commonly employed in the literature of heredity) a "gamete" of the normal ivory-coloured stock will here be denoted by the letters *fE*, and a gamete of the red peloric stock by the letters *Fe*. In this notation, the individual letters have the following significance:

F capacity for forming red flower-pigment	*f* lack of such capacity; i.e. the gamete thus designated transmits the ivory flower tint
E capacity for forming flowers of the normal snapdragon shape	*e* lack of such capacity; i.e. this gamete transmits the peloric flower shape

One P_1 plant, originating out of the union of gametes *Fe* (red flower-pigment and peloric blossoms), will then have the formula *FFee*.

The other P_1 plant, originating out of the union of gametes *fE* (ivory flowers of normal snapdragon shape), will have the formula *ffEE*.

The hybrid, therefore, will have the formula *FfEe*. When this hybrid becomes sexually mature, it will according to the mendelian theory form four different kinds of reproductive cells or gametes, as follows:

FE transmitting the red colour and the normal shape of flower
Fe ,, ,, red ,, ,, ,, peloric ,, ,, ,,
fE ,, ,, ivory ,, ,, ,, normal ,, ,, ,,
fe ,, ,, ivory ,, ,, ,, peloric ,, ,, ,,

These four kinds of gametes will be formed in equal numbers.

If we allow self-fertilisation to occur in such a hybrid or allow a number of such hybrids to fertilise one another, sixteen different combinations can occur among the four kinds of ovules and the four kinds of pollen grains, as follows:

1. an ovule *FE* can encounter a pollen grain *FE* and produce a red normal plant *FFEE*
2. ,, ,, *FE* ,, ,, ,, ,, ,, *Fe* and produce a red normal plant *FFEe*
3. ,, ,, *FE* ,, ,, ,, ,, ,, *fE* and produce a pink normal plant *FfEE*
4. ,, ,, *FE* ,, ,, ,, ,, ,, *fe* and produce a pink normal plant *FfEe*
5. ,, ,, *Fe* ,, ,, ,, ,, ,, *FE* and produce a red normal plant *FFEe*
6. ,, ,, *Fe* ,, ,, ,, ,, ,, *Fe* and produce a red peloric plant *FFee*
7. ,, ,, *Fe* ,, ,, ,, ,, ,, *fE* and produce a pink normal plant *FfEe*
8. ,, ,, *Fe* ,, ,, ,, ,, ,, *fe* and produce a pink peloric plant *Ffee*
9. ,, ,, *fE* ,, ,, ,, ,, ,, *FE* and produce a pink normal plant *FfEE*
10. ,, ,, *fE* ,, ,, ,, ,, ,, *Fe* and produce a pink normal plant *FfEe*
11. ,, ,, *fE* ,, ,, ,, ,, ,, *fE* and produce an ivory normal plant *ffEE*
12. ,, ,, *fE* ,, ,, ,, ,, ,, *fe* and produce an ivory normal plant *ffEe*
13. ,, ,, *fe* ,, ,, ,, ,, ,, *FE* and produce a pink normal plant *FfEe*
14. ,, ,, *fe* ,, ,, ,, ,, ,, *Fe* and produce a pink peloric plant *Ffee*
15. ,, ,, *fe* ,, ,, ,, ,, ,, *fE* and produce an ivory normal plant *Effe*
16. ,, ,, *fe* ,, ,, ,, ,, ,, *fe* and produce an ivory peloric plant *ffee*

A glance at the foregoing table shows that of the sixteen possible conjugations, three (1, 2, 5) must produce plants with red normal flowers, six (3, 4, 7, 9, 10, 13) plants with pink normal flowers, one (6) plants with red peloric flowers, two (8, 14) plants with pink peloric flowers, three (11, 12, 15) plants with ivory normal flowers, and one (16) plants with ivory peloric flowers.

In F_2 of this crossing we ought to find specimens of the six different kinds of plant—red normal, pink normal, red peloric, pink peloric, ivory normal, ivory peloric—in the ratios 3 : 6 : 1 : 2 : 3 : 1. If (as in Fig. 9) we count reds and pinks

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together, the ratios will be 9 : 3 : 3 : 1. In actual experiments, the ratios obtained harmonise closely with those deducible by the mendelian theory. For instance, as an outcome of one such crossing the F_2 generation consisted of the following:

Red normals, 39; pink normals, 94; red pelorics, 15; pink pelorics, 28; ivory normals, 45; ivory pelorics, 13.

red peloric × ivory normal P_1
pink normal F_1
F_2

red normal $\frac{3}{16}$	pink normal $\frac{6}{16}$	red peloric $\frac{1}{16}$	pink peloric $\frac{2}{16}$	ivory normal $\frac{3}{16}$	ivory peloric $\frac{1}{16}$
one part (No. 1) remains constant red normal, two parts (Nos. 2, 5) separate into 3 red normal to 1 red peloric	two parts (Nos. 3, 9) separate into $\frac{1}{4}$ ivory normal, $\frac{2}{4}$ pink normal, $\frac{1}{4}$ red normal; four parts (Nos. 4, 7, 10, 13) separate exactly like the original hybrid into $\frac{3}{16}$ red normal, $\frac{6}{16}$ pink normal, $\frac{1}{16}$ red peloric, $\frac{2}{16}$ pink peloric, $\frac{3}{16}$ ivory normal, $\frac{1}{16}$ ivory peloric.	remains constant	separate into $\frac{1}{4}$ ivory peloric, $\frac{2}{4}$ pink peloric, $\frac{1}{4}$ red peloric	two parts (Nos. 12, 15) separate into $\frac{1}{4}$ ivory peloric, $\frac{3}{4}$ ivory normal, one part (No. 11) remains constant	remains constant

On the basis of the theory we must further expect that one third of the normally flowering categories will have offspring producing exclusively normal flowers, this being the case with all the plants which contain *EE* (Nos. 1, 3, 9, 11); and we must expect that two thirds of them, namely all those which contain *Ee* (Nos. 2, 4, 5, 7, 10, 12, 13, 15), will subsequently separate into normal and peloric offspring; and we must also expect that all the red *FF* plants will have exclusively red offspring, whereas all the pink *Ff* plants will in the next generation exhibit a separation into red, pink, and ivory plants. These theoretical expectations are fulfilled in all the experiments. Such an experiment is shown on p. 52 in the form of a genealogical tree.

Thus we see that the various characters by which the races originally crossed one with another are distinguished from one another must be separately and independently distributed to the reproductive cells.

This law of the independent hereditary transmission of the individual characters by which the stocks originally crossed are distinguished one from another is of the greatest importance in the study of the processes of inheritance in the human species likewise.

A crossing which corresponds in every respect to the one just described, but in which both the characters are dominant, is shown in Fig. 10. It represents the crossing of a smooth-haired black guineapig race with a rough-haired white guineapig race. In F_1 we get rough-haired black animals, and in F_2 the four combinations to be expected on the mendelian theory make their appearance, for we have black rough-haired, white rough-haired, black smooth-haired, and white smooth-haired guineapigs, in the ratios 9 : 3 : 3 : 1.

We can cross races which differ from one another in respect of more numerous characteristics. For instance, we can cross an ivory normal tall snapdragon with a red peloric short snapdragon. Then, F_1 is red, normal, and tall. Speaking generally, tallness is a dominant character. It we call a gamete which transmits the character of "tallness" *X*, and one which transmits the character of "shortness" *x*, while retaining for shape and colour the previous designations, then the "formula" of the ivory normal talls will be *ff EE XX*, and that of the red peloric shorts will be *FF ee xx*. The hybrid will be *Ee Ff Xx*, and will form gametes of 8 different kinds, namely *FEX*, *FEx*, *FeX*, *Fex*, *fEX*, *fEx*, *feX*, *fex*, among which 64 different kinds of conjugation are possible. But these 64 combinations likewise are not all outwardly different. If we leave out of account the differences between the homozygotes and the heterozygotes, we find in F_2 eight different kinds of plants, namely: 1. red normal tall (27); 2. red normal short (9); 3. red peloric short (9); 4. red peloric tall (9); 5. ivory normal tall (3); 6. ivory normal short (3); 7. ivory peloric tall (3); 8. ivory peloric short (1). In each case the number of the

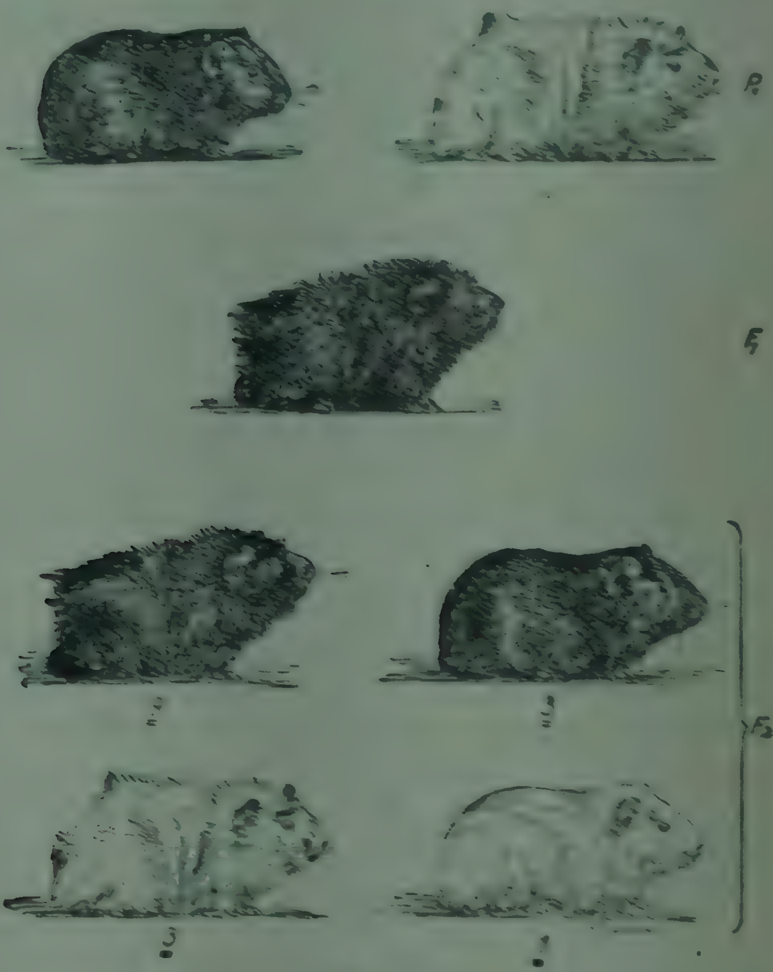

FIG. 16.

Crossing of a black smooth-haired guineapig race with a white rough-haired race. The F_1 animals are black, rough-haired beasts, while these in the F_2 generation are black rough-haired, black smooth-haired, white rough-haired, and white smooth-haired beasts, in the ratios 9 : 3 : 3 : 1.

different kinds of plants in the F_2 generation is shown by the figures in parenthesis, which thus give the ratios of the different kinds. Each of these types can be bred homozygotically, so that it is easy, as the outcome of one such crossing of stocks, to originate a number of new stocks, which, however, are substantially nothing more than new combinations among the characters independently transmissible by inheritance in the original stocks or races. If we cross races which differ from one another in a larger number of characters, the same laws apply. The following table (p. 56) shows the numerical ratios that will then be exhibited, and gives a general formula for *n* characters.

Of course, with an increase in the number of independent inheritable differences between two crossed races, the composition of the F_2 generation becomes extraordinarily complicated. If, for example, the two races have 10 independent differences, there will in F_2 be $2^{10} = 1024$ visibly different kinds of individuals.

A great many of the results of crossing which at the outset do not seem to conform to the laws of the separation or independent assortment of characters, are found on closer study to be in perfect harmony with these laws. No doubt the phenomena of separation are complicated in various ways which are apt to make it difficult to understand exactly what has happened in a crossing. The most important of these complications is that an apparently unified quality or character always depends upon several independent mendelian "factors". For instance, the brown pigment in a bird's feathers may originate in this way, that a colourless chromogen develops the brown pigment only when it is acted upon by an enzyme. But the "capacity for forming the enzyme" and the "capacity for forming the chromogen" may be separately transmissible and mutually independent mendelian characters. For instance, in a white breed of fowls the capacity for the formation of the enzyme may be lacking when the capacity for the formation of the chromogen is present. In another white breed, the capacity for the formation of the chromogen may be lacking when the capacity for the formation of the enzyme is present. If we cross two such white breeds in which the whiteness is

Number of the differences between the P_1 individuals.	Number of the different kinds of gametes formed in F_1.	Number of the possible combinations of gametes.	Maximum number of the outwardly distinguishable categories of F_2 individuals when complete dominance prevails throughout.	The outwardly distinguishable kinds of F_2 individuals are, when complete dominance prevails throughout, represented by figures which bear the following ratios to one another. (If an outwardly distinguishable character is dependent upon the presence of several heterozygotic factors, different figures will apply, but will be deducible from those here given.)
1	2^1 $= \mathbf{2}$	$(2^1)^2$ $= \mathbf{4}$	2^1 $= \mathbf{2}$	$\underbrace{3}_{1} : \underbrace{1}_{1}$
2	2^2 $= \mathbf{4}$	$(2^2)^2$ $= \mathbf{16}$	2^2 $= \mathbf{4}$	$\underbrace{9}_{1} : \underbrace{3 : 3}_{2} : \underbrace{1}_{1}$
3	2^3 $= \mathbf{8}$	$(2^3)^2$ $= \mathbf{64}$	2^3 $= \mathbf{8}$	$\underbrace{27}_{1} : \underbrace{9 : 9 : 9}_{3} : \underbrace{3 : 3 : 3}_{3} : \underbrace{1}_{1}$
4	2^4 $= \mathbf{16}$	$(2^4)^2$ $= \mathbf{256}$	2^4 $= \mathbf{16}$	$\underbrace{81}_{1} : \underbrace{27 : 27 : 27 : 27}_{4} : \underbrace{9 : 9 : 9 : 9 : 9 : 9}_{6} : \underbrace{3 : 3 : 3 : 3}_{4} : \underbrace{1}_{1}$
n	2^n	$(2^n)^2$	2^n	$\underbrace{3^n}_{1} : 3^{n-1} : 3^{n-1} : 3^{n-1} \dots 3^{n-2} : 3^{n-2} : 3^{n-2} \dots$ etc. . . . etc. = coefficients of the binomial $(a + a)^n$ . . . 1

due to different causes, we shall get hybrids inheriting from one parent the capacity for the formation of the enzyme and from the other the capacity for the formation of the chromogen, and the resulting birds will, owing to "hybrid atavism", have pigmented feathers. If we call the heredity factor which renders the formation of the chromogen possible A, and the heredity factor which renders possible the formation of the enzyme B, then a white breed which can form the chromogen only will be AA, bb, and a white breed which can form the enzyme only will be $aa\ BB$. The hybrids formed from these two breeds will then be $Aa\ Bb$, will be capable of forming both chromogen and enzyme, and will therefore have pigmented feathers. These hybrids $Aa\ Bb$ produce gametes of four different kinds AB, Ab, aB, ab, and among these gametes 16 combinations are possible. As the following table of combinations shows, 9 of the combinations will produce birds with pigmented feathers and 7 will produce birds with white feathers.

$AB \times AB = AABB$ pigmented
$AB \times Ab = AABb$,,
$AB \times aB = AaBB$,,
$AB \times ab = AaBb$,,

$Ab \times AB = AABb$ pigmented
$Ab \times Ab = AAbb$ white
$Ab \times aB = AaBb$ pigmented
$Ab \times ab = Aabb$ white

$aB \times AB = AaBB$ pigmented
$aB \times Ab = AaBb$,,
$aB \times aB = aaBB$ white
$aB \times ab = aaBb$,,

$ab \times AB = AaBb$ pigmented
$ab \times Ab = Aabb$ white
$ab \times aB = aaBb$,,
$ab \times ab = aabb$,,

If we make a series of crossing experiments with any plant or animal, we shall soon be able to detect the existence of a number of such heredity factors, and it will again and again become manifest that the apparently vast number of differences of race, breed, or stock within a particular "species" are nothing more than varying combinations among a comparatively small number of mendelian differences. This is usually expressed more precisely by saying that they are the result of a comparatively small number of "heredity factors". This can be shown most easily in the case of rabbits. The commoner breeds of rabbit, as distinguished by the colour of the fur, are determined by the following heredity factors:

Heredity factor	*Effect*
A a	None of the *aa* rabbits can form pigment in the skin or the eyes, no matter what their heredity formula may be in other respects, and they are therefore white with red (i.e. pigmentless) eyes—typical albinos. *AA* and *Aa* rabbits, lacking the second important pigment-factor *X*, and therefore having *xx* in their heredity formula, are white rabbits with blue eyes—for instance the "white Viennese rabbits".
X x	*X* is the second main factor in pigment-formation, and in conjunction with *A* it renders possible the growth of fur with a yellowish tint.
B b	When this factor is present, the yellow tint due to *A* and *X* undergoes intensification into light brown.
C c	*C* transforms into blue the brown tint achieved by *A*, *X*, and *B*.
D d	*D* makes the fur still darker, deepening the tint due to the foregoing factors. *AXBcd* is light brown. *AXBcD* is chocolate brown. (This is the colour of the rabbits described as "Havana-coloured" or "nigger-brown.") *AXBCd* is blue (like the "blue Viennese"). *AXBCD* is a rich black.
G g	*G* has the effect of making the hair particoloured, showing alternate zones of lighter and darker pigmentation. A rabbit having the formula *AXBCDg* is black; one having the formula *AXBCDG* is "wild-coloured", i.e. its fur has the same tint as that of the wild rabbit. In like manner, a rabbit having the formula *AXBCdg* is uniformly blue; one having the formula *AXBCdG*, on the other hand, "blue-wild-coloured", i.e. blue in fundamental tone, but with the particoloured or zonular distribution of hair-pigment characteristic of the wild rabbit. Similarly among the yellow rabbits there may be some that are yellow-wild-coloured; and so on. Apart from its general effect upon the tint of the fur, *G* also manifests its influence in this, that all *G* rabbits have a white or very light-tinted belly, with the under side of the tail likewise white, whereas in all *gg* rabbits the belly and the under side of the tail are as deeply tinted as other parts of the fur.
K k	All *K* rabbits are spotted, inasmuch as there are irregular white patches in a fur which is in other respects tinted as determined by the general formula.
M m	All *mm* rabbits whose formula in other respects would

Heredity factor	*Effect*
	make them black, have black patches on a yellow ground. In like manner all *mm* rabbits whose formula would in other respects make them wild-coloured, have wild-coloured patches upon a yellow wild-coloured ground; and all those whose formula would in other respects make them blue, have blue patches on a light-blue ground; and so on.
N n	The characteristic of *nn* rabbits is that the colour which their formula in other respects determines appears only on the muzzle, ears, paws, and tail. Animals thus tinted are said to be "soot-coloured". Except for the markings, their fur is white, and they have red eyes. The colour of the "peripheral" markings is determined by the other elements of their formula. Thus there are wild-coloured, black, blue, etc., "soots". This factor *N* is "unilocal" with *A* (see below, p. 80).
O o	*O* likewise determines a particular distribution of colour. A rabbit having the formula *AXBCDgkMNo* is pure black; one having the formula *AXBCDgkMNO* is black and tan, the tan patches appearing above the eyes, on the ears, on the paws, and elsewhere—the general design being that often seen in dachshunds, Dobermann terriers, etc. *O* rabbits whose formula in other respects would make them blue, are blue and tan; and so on.
P p	All *P* rabbits, whatever their general tint as determined by their formula in other respects, are "greyed" or "silvered" by the random sprouting of white hairs. This silvering, which obviously corresponds to the greying of horses, only begins to appear when the rabbit is about six weeks old. It depends upon several factors working in the same direction and mutually reinforcing one another (see below, pp. 61-3).
V v	All *vv* rabbits have Angora fur.

These twelve fundamental differences, variously combined, render it possible to produce a very large number (4096) of breeds; and by the crossing of rabbits whose formulas are known we can make any new combination we please, thus producing new breeds, as can be shown by an example.

A rabbit having the formula *aa XX BB CC DD gg kk MM NN oo pp vv* is a white Angora with red eyes. A rabbit with the formula *AA XX BB CC dd GG kk MM NN oo pp VV* is blue-wild-coloured and short-haired.

The hybrids derived from these two animals are wild-coloured and smooth-haired, being thus exactly like wild rabbits. In F_2, however, a separation occurs into ten outwardly distinguishable kinds: 1. wild-coloured short-haired; 2. wild-coloured Angora-haired; 3. black short-haired; 4. black Angora-haired; 5. blue wild-coloured short-haired; 6. blue wild-coloured Angora-haired; 7. blue-gray short-haired; 8. blue-gray Angora-haired; 9. white short-haired; 10. white Angora-haired. Or, to give another example, the crossing of *aa XX BB CC dd gg kk MM NN oo pp vv* (a white Angora rabbit) with an *AA xx BB CC dd GG kk MM NN oo pp VV* (a white Viennese rabbit) produces wild-coloured short-haired rabbits which subsequently in F_2 produce extremely varied offspring assorted in accordance with the formula. We may compare this with the result of the crossing of an albino negro (whose skin is white) with a fair European, when the offspring belonging to F_1 are as dark as ordinary mulattoes.

The before-mentioned fundamental differences are not the only ones hitherto worked out in the case of rabbits. We know to-day of more than fifty such differences, but their number is small in comparison with the immense number of different breeds. The twelve fundamental differences already considered render possible, as explained, 4096 different hereditary types, of which, indeed, $\frac{1}{4}$, that is to say 1024, are white rabbits with red eyes, while another 768 are white rabbits with blue eyes. But all the other 2304 types are outwardly distinguishable. Although only a small number of these have at the present time actually been bred for the market, it is easy to make combinations standing outside the general run and to make pure "breeds" of them. As soon as the characters of a species of animal have been sufficiently analysed, we can deal with it synthetically just as a chemist deals with chemical compounds, and can deliberately produce any desired combination of characters—that is to say, we can originate new breeds.

That which in a finished organism has the aspect of a unitary character—a particular tint of hair, for instance—will be found, as we have already learned from numerous examples, to be determined by a considerable number of heredity factors. Consider, for instance, the factors which cooperate in producing the wild colour of rabbits. It constantly happens, too, that distinct factors, which are independently transmitted by inheritance, manifest themselves in identical ways. An example will illustrate this. One variety of wheat may be fairly immune to parasitic fungi because the structure of its cell walls affords an especially good protection against the invasion of the fungi. Another variety of wheat may have the same degree of immunity though in this case the immunity depends upon the presence of particular protective substances in the cell juice. If we cross these two varieties of wheat endowed with an equal degree of immunity and perhaps not outwardly distinguishable, we shall get a hybrid inheriting both the causes of immunity but containing them only heterozygotically, and therefore (if no more than partial dominance exists) immune to approximately the same degree as each of the parent varieties. In the F_2 generation of these hybrids, however, there will be plants which have homozygotically inherited both the causes of the immunity, and therefore will be twice as immune as was either of the parent varieties. Furthermore, since there will likewise be recessive combinations, there will in this generation be some plants which will have inherited neither of the two causes of immunity, and will therefore no longer be immune. Finally, there will be a number of intermediate types whose immunity will be approximately equal to that of the parent races. A very instructive instance of this may be given, borrowed from Nilsson-Ehle, to whom we owe so much of the light which has been thrown on the previously incomprehensible phenomena of inheritance. (It is probable that similar relationships occur frequently among human beings.) In wheat, the reddish colour of the grain is due to at least three mutually independent mendelian factors, which we will term *R*, *S*, and *T*. A plant which heterozygotically contains only one of these factors—an *Rr ss tt* plant, that is to say, or an *rr Ss tt* plant, etc.—is of a pale reddish tint; a plant which contains one of these factors homozygotically—an *RR ss tt*, or an *rr SS tt*, etc. —is of a rather darker reddish colour; and, finally, at the other end of the scale, plants which contain all three factors homozygotically—*RR SS TT*—exhibit the darkest of the reddish tints.

If we cross a white race of wheat containing not one of the

Possible Combinations of Gametes	F_2	F_3
1. RST × RST	red VI	red[1]
2. RST × RSt	,, V	,,
3. RST × RsT	,, V	,,
4. RST × Rst	,, IV	,,
5. RST × RST	,, V	,,
6. RST × rSt	,, IV	,,
7. RST × rsT	,, IV	,,
8. RST × rst	,, III	separating into 63 reds : 1 white
9. RSt × RST	,, V	red
10. RSt × RSt	,, IV	,,
11. RSt × RsT	,, IV	,,
12. RSt × Rst	,, III	,,
13. RSt × rST	,, IV	,,
14. RSt × rSt	,, III	,,
15. RSt × rsT	,, III	separating into 63 reds : 1 white
16. RSt × rst	,, II	,, ,, ,, 15 ,, : 1 ,,
17. RsT × RST	,, V	red
18. RsT × RSt	,, IV	,,
19. RsT × RsT	,, IV	,,
20. RsT × Rst	,, III	,,
21. RsT × rST	,, IV	,,
22. RsT × rSt	,, III	separating into 63 reds : 1 white
23. RsT × rsT	,, III	constant
24. RsT × rst	,, II	separating into 15 reds : 1 white
25. Rst × RST	,, IV	red
26. Rst × RSt	,, III	,,
27. Rst × RsT	,, III	,,
28. Rst × Rst	,, II	,,
29. Rst × rST	,, III	separating into 63 reds : 1 white
30. Rst × rSt	,, II	,, ,, 15 ,, : 1 ,,
31. Rst × rsT	,, II	,, ,, 15 ,, : 1 ,,
32. Rst × rst	,, I	,, ,, 3 ,, : 1 ,,
33. rST × RST	,, V	red
34. rST × RSt	,, IV	,,
35. rST × RsT	,, IV	,,
36. rST × Rst	,, III	separating into 63 reds : 1 white
37. rST × rST	,, IV	red
38. rST × rSt	,, III	,,
39. rST × rsT	,, III	,,
40. rST × rst	,, II	separating into 15 reds : 1 white

---

[1] "Red" signifies here that no white plants separate out; but in other respects the significance of the term is not homogeneous, for these reds separate, according to the formula in each case, into light-reds and dark-reds respectively.

Possible Combinations of Gametes	F_2	F_3
41. rSt × RST	red IV	red
42. rSt × RSt	,, III	,,
43. rSt × RsT	,, III	separating into 63 reds : 1 white
44. rSt × Rst	,, II	,, ,, 15 ,, : 1 ,,
45. rSt × rST	,, III	red
46. rSt × rSt	,, II	,,
47. rSt × rsT	,, II	separating into 15 reds : 1 white
48. rSt × rst	,, I	,, ,, 3 ,, : 1 ,,
49. rsT × RST	,, IV	red
50. rsT × RSt	,, III	separating into 63 reds : 1 white
51. rsT × RsT	,, III	red
52. rsT × Rst	,, II	separating into 15 reds : 1 white
53. rsT × rST	,, III	red
54. rsT × rSt	,, II	separating into 15 reds : 1 white
55. rsT × rsT	,, II	red
56. rsT × rst	,, I	separating into 3 reds : 1 white
57. rst × RST	,, III	,, ,, 63 reds : 1 ,,
58. rst × RSt	,, II	,, ,, 15 ,, : 1 ,,
59. rst × RsT	,, II	,, ,, 15 ,, : 1 ,,
60. rst × Rst	,, I	,, ,, 3 ,, : 1 ,,
61. rst × rST	,, II	,, ,, 15 ,, : 1 ,,
62. rst × rSt	,, I	,, ,, 3 ,, : 1 ,,
63. rst × rsT	,, I	,, ,, 3 ,, : 1 ,,
64. rst × rst	white O	white

three factors (*rr ss tt*) with a homozygotically dark-red wheat (*RR SS TT*) we get a hybrid (*Rr Ss Tt*) which has a medium tint and is therefore pale red. This hybrid forms 8 kinds of gametes, *RST*, *RSt*, *RsT*, *Rst*, *eST*, *rSt*, *rsT*, *rst*, and these 8 kinds of gametes can conjugate in the 32 different ways shown in the foregoing table.

The second column of the table shows the tint of the grain in the respective categories. Here the darkest red tint, due to the presence of the three factors homozygotically, is denoted "red VI"; white is denoted "red O"; the palest of the reddish tints, due to a single heterozygotic factor, is termed "red I"; the intermediate grades are II to V.

The table shows that, in the F_2 generation of the crossing, there must appear side by side with the quite exceptional plants which exactly reproduce the colouring of the parental generation, a long series of diversely coloured intermediate stages, that is to say "reds" in which the red is variously graded from I to V.

If we examine the table to ascertain the frequency of occurrence of the various tintings in F_2, we find:

1	combination of plants with the colour red	VI	(like one of the parent plants)
6	combinations ,, ,, ,, ,, ,, ,,	V	
15	,, ,, ,, ,, ,, ,, ,,	IV	
20	,, ,, ,, ,, ,, ,, ,,	III	
15	,, ,, ,, ,, ,, ,, ,,	II	
6	,, ,, ,, ,, ,, ,, ,,	I	
1	combination ,, ,, ,, ,, ,, ,,	O	(white, like the other parent plant)

We must expect, therefore, that F_2 of such a crossing will consist of plants the great majority of which will exhibit an average strength of the red tints, while there will be a few of a very dark red, and also a few that are very light in colour. Furthermore, a glance at the figures 1, 6, 15, 20, 15, 6, 1 which represent the frequency with which the various intensities of reddening appear, shows that the series corresponds to the curve of probability.

We see, therefore, that when, as in our example, the colour of the wheat grain is influenced by a number of independent mendelian factors, which, though they operate independently of one another, act in the same direction so as to produce a joint effect, then a plant equipped with several of these factors heterozygotically will have offspring displaying a series of gradations of colour. Among these gradations, plants with a mean intensity of colour will be frequent, whereas very light and very dark plants will be much less common, becoming continually more and more infrequent, in accordance with the curve of probability, in proportion as they are lighter or darker as the case may be.

This is the probable explanation of most of the instances of crossing recorded in the literature of the subject concerning which we are told that the hybrids occupied an intermediate position between their parents, and in subsequent generations did not separate out, but continued to transmit the intermediate characters constantly by inheritance. For instance, as regards the tint of skin when Negroes are crossed with whites, we are told that the resulting mulattoes have offspring whose intensity of colour remains constant for generations. But according to the more carefully conducted recent investigations, this is not the case. From a union between two F_1 mulattoes there may even, though in very rare instances, be born pure whites and pure blacks; but the great majority of the offspring of such unions have, approximately, the tint of the F_1 mulattoes. If we assume that the difference in the colour of the skin as between Negroes and white Europeans is, like the colour of the wheat grains in the foregoing example, determined by the joint action of several independent mendelian factors, it is perfectly comprehensible why the result of breeding from mulattoes should be that the enormous majority of the offspring have approximately the same tint as the parents, while in exceptional cases children are born having the black tint or the white tint of the primary parental stocks.

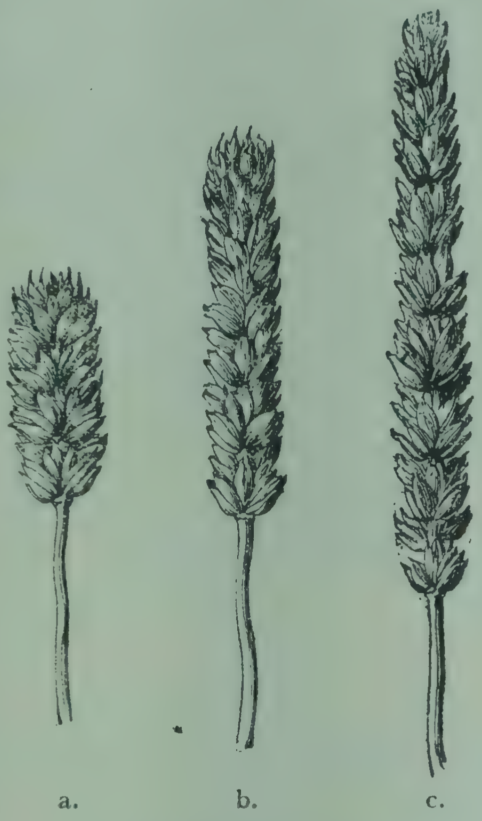

FIG. 11.
Shape of the ear in wheat.
a. Compactum wheat.
b. Squarehead wheat.
c. Loose-eared wheat of ordinary cultivation.

We shall especially often be able to detect the joint working of independent factors when we study inheritance in the mass. How remarkable the phenomena of inheritance may be in such cases is illustrated once more by the account of an experiment on wheat. The length of the ear in wheat is influenced by a considerable number of heredity factors. First of all, we know of at least two jointly acting factors which make the ears long and loose. Let us call these two factors *L* and *M*. A plant having the formula *LL MM* has a very long and loose ear (c, in Fig. 11); all plants containing one of the two factors, either *ll MM* or *LL mm*, have a medium length of ear; and, finally a plant having the formula *ll mm* is a "squarehead" (b, in Fig. 11). Another important factor, *C*, makes the ears very short and compressed (a, in Fig. 11). *C* is dominant over *L* and *M*, so that there are plants having the formula *CC LL MM*, or *CC ll MM*, or *CC ll mm*, or *Cc LL MM*, etc., which are not outwardly distinguishable from compactum wheat. These considerations enable us to understand many very remarkable results of crossing. First, for instance, the crossing of two varieties of wheat both having an ear of medium length will sometimes produce a hybrid also having an ear of medium length, but in the F_2 generation the working of the mendelian factors will lead to the appearance, side by side with a great many plants having ears of medium length, of some plants having very long and loose ears and of other plants having short ears (squarehead wheat). In this case the crossing will have taken place as follows:

cc LL mm medium length	×	*cc ll MM* medium length	P_1
	cc Ll Mm medium length		F_1

In F_2, etc. the working of the mendelian factors must likewise result in the production of *cc LL MM* (wheat with long, loose ears) and *cc ll mm* (squarehead wheat).

Further, the crossing of *CC LL MM* (compactum) with *cc ll mm* (squarehead) must in F_1 produce only compactum plants (*Cc Ll Mm*); but in F_2 there will occur a separation into compactum, long-eared, medium long-eared, and squarehead wheat.

Any one who is investigating the laws of the hereditary transmission of racial differences in man, such as the transmission of the shape of the skull and similar matters, should continually bear in mind these clear and luminous examples from the vegetable kingdom. When F_1 consists of two races one of which has a fairly dominant character, we seldom find that in F_2 there is a perfectly simple separation, that is to say a separation in accordance with this one factor. Certain conspicuous malformations and other errors of development, such as radial flowers in snapdragon (Antirrhinum—see Fig. 9), brachydactylia or abnormal shortness of the fingers and toes in human beings, albinism or complete absence of pigment in human beings and many animals, bowed fore-legs in many mammals, etc., are, as a rule, determined by one heredity factor only, and their transmission is therefore perfectly simple; but racial differences, and above all the differences between "wild" or "savage" races, in plants, animals, and human beings, usually depend upon several different heredity factors, and often upon an astonishingly large number of these. In the human species, indeed, most of the more striking malformations and errors of development depend upon single heredity factors, and their course can therefore be easily traced in successive generations. On the other hand, differences in the shape of the skull, the structure of the brain, the shape of the nose, the formation of the teeth, the colour of the eyes, the colour of the skin, etc., etc.—in a word, the numberless morphological and physiological distinctions between the various races of man—depend in each case upon very numerous heredity factors, with the result that as yet we know little about the course of their inheritance.

In man, we cannot study heredity by the experimental method, and have to be content with the analysis of genealogical trees. One who wishes to obtain detailed information concerning the hereditary transmission of a morbid predisposition or of some other striking characteristic which recurs for several generations, can only do so by securing genealogical trees, as many as possible and as complete as possible, from the families thus affected. Often enough, the first glance at a genealogical tree of this kind will show whether the family peculiarity is or is not determined by a single factor, and whether we have to do with a recessive or a dominant character. Let us suppose that, in the case of some such transmissible defect as a rupture or a hammer-toe, the family history enables us to draw the genealogical tree shown in Fig. 12. Unquestionably, in this case, the hereditary defect is dominant and unifactoral. If, in the case of another hereditary defect, such as albinism, we get the genealogical tree shown in Fig. 13, then we have to do with a recessive unifactoral hereditary defect.

Such genealogical trees, however, are far from always being so simple and so easy to interpret. Difficulty arises especially in those cases in which a predisposition can be transmitted without necessarily becoming manifest in the individual who inherits it. For example, a predisposition to rupture is transmitted according to simple laws, but not every individual who inherits the liability develops a rupture. When we study the ancestry of individuals affected with hernia, we may find that for several generations back there has been no case of rupture in their families. We should, however, be quite wrong to infer from this that we have to do here with a recessive malformation.

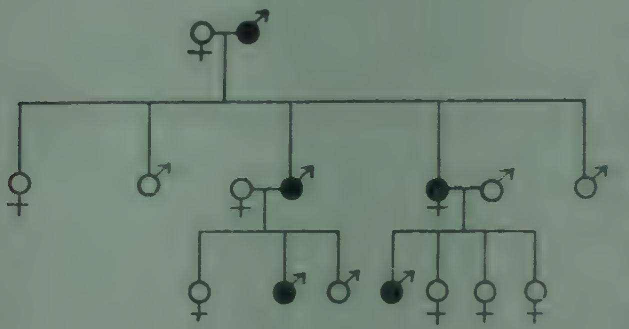

FIG. 12.[1]

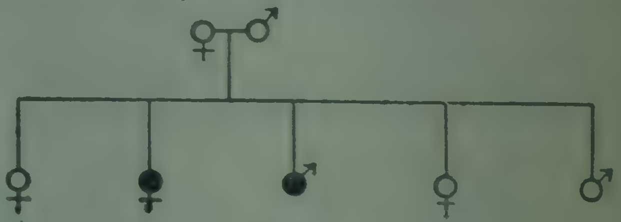

FIG. 13.

Further, it often happens that ostensibly identical malformations, like the character "whiteness" in the white breed of fowls described above on pp. 55 et seq., may be genetically entirely different. These complications, and many others, must all be taken into account when we are interpreting family trees.

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[1] Black circles indicate diseased individuals; white circles, healthy individuals. The arrow pointing upwards denotes a male, the cross pointing downwards denotes a female.

b. THE COUPLING OF FACTORS

Any one who makes experiments in crossing, such experiments as those previously described, no matter with what organism, will sooner or later come across the phenomenon which is known as the coupling of factors.

Let us begin our consideration of this matter, once more, with a very simple instance. The small fruit-fly, Drosophila melanogaster, is the animal organism which at the present day has been most exhaustively studied in respect of its heredity factors. Several hundred heredity factors have already been clearly distinguished in its case. For the moment, however, we shall consider only two of these, the factors *B* and *V*.

A race which contains both of these factors, together with all the other factors requisite for normal bodily structure, looks like an ordinary wild Drosophila. A race having in other respects the same formula, but *bb*, is pure black (see the left upper part of Fig. 14); a race with *vv* has stumpy wings, (i.e. the wings are rudimentary—see the upper right corner of Fig. 14). The crossing of these two races gives in F_1, fruit-flies having the formula *Bb Vv* which are wild-coloured and normal-winged (see Fig. 14, F_1).

What we have previously learned shows that such hybrids, *Bb Vv*, ought to produce four kinds of offspring, *BV*, *Bv*, *bV*, and *bv*—the four kinds occurring with equal frequency.

Furthermore, from what we have already learned, we must expect that when a hybrid female *Bb Vv* is recrossed with a doubly recessive male *bb vv* (black and stumpy-winged), four kinds of insects will be produced: 1. wild-coloured normal-winged; 2. black normal-winged; 3. wild-coloured stumpy-winged; and 4. black stumpy-winged—and that all these four kinds will appear with equal frequency. Let us set the matter out in a table:

ovum *BV* encounters spermatozoid *bv*, producing *Bb Vv*
= wild-coloured normal winged.
,, *Bv* ,, spermatozoid *bv*, producing *Bb vv*
= wild-coloured stumpy-winged.
,, *bV* ,, spermatozoid *bv*, producing *bb Vv*
= black normal-winged.
,, *bv* ,, spermatozoid *bv*, producing *bb vv*
= black stumpy-winged.

These four types of insect (Fig. 14, F_2) are actually produced in the experiment, but they do not appear in the equal numbers we have been led to expect. Instead of this, the various kinds are produced in approximately the following ratios:

wild-coloured	normal-winged	1
,, ,,	stumpy-winged	5
black	normal-winged	5
,,	stumpy-winged	1

These results lead us to infer that the F_1 females must produce their four categories of ova *BV*, *Bv*, *bV*, *bv*, in this same ratio 1 : 5 : 5 : 1. In other words, these F_1 females form the two gamete types out of whose union they have themselves originated (in the instance we are now considering *bV* and *Bv*) more frequently than the two gamete types wherein the heredity

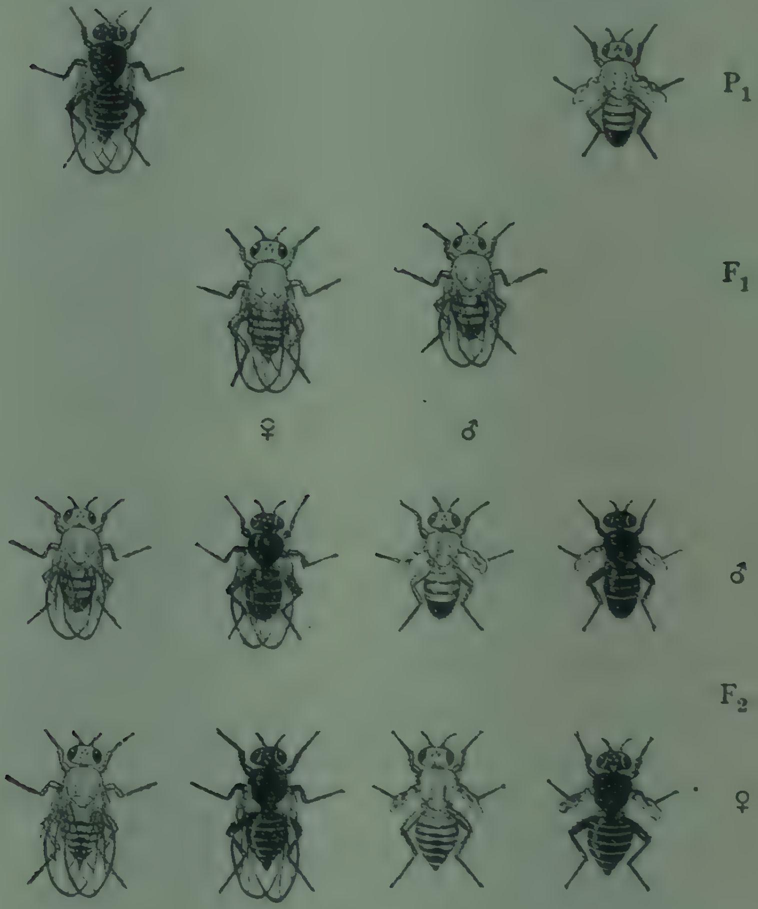

FIG. 14.

factors are contained in new combinations (in the instance we are considering *BV* and *bv*).

The same rule applies when we produce a hybrid *Bb Vv* in the other way that is possible (Fig. 15). Our first hybrid was produced by the crossing of black normal-winged with wild-coloured stumpy-winged. But we can produce just such another hybrid by crossing *bb vv* with *BB VV* (Fig. 15). F_1 is then likewise wild-coloured normal-winged; and when we now proceed, as before, to cross an F_1 female with a doubly recessive male *bb vv*, we get offspring in which the expected four types occur, but now in the ratio:

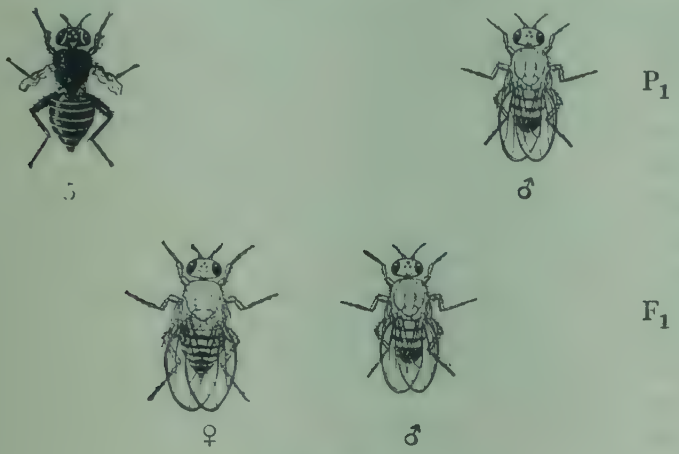

Fig. 15.

wild-coloured normal-winged	5
,, ,, stumpy-winged	1
black normal-winged	1
,, stumpy-winged	5

In this instance likewise, the hybrid female must have formed ova having the original combination of factors (here *BV* and *bv*) five times as frequently as the ova with the new combination (in this case *Bv* and *bV*).

Such instances of the coupling or linkage of factors have been very frequently encountered both in plants and in animals. For the study of human heredity, too, this matter is of great interest, and it is of the utmost theoretical importance as concerns our whole way of contemplating the cytological basis of mendelian separation and of racial differences in general.

This question must, therefore, be briefly considered here. I have already pointed out that every hereditary racial distinction must, in the last resort, depend upon some difference or other in the chemical composition or the minute structure of the idioplasm.

We can to-day assert with the utmost confidence that for the most part the idioplasm (for the time being this is to us no more than a theoretical concept deduced from the doctrine of heredity) must be localised in the cell nucleus, and the researches of recent years make it extremely probable that a mendelian hereditary difference is referable to a difference between two homologous chromosomes. I assume that my readers know, but will here remind them of, the elementary fact that in all organisms reproducing themselves sexually every gamete or reproductive cell has a single or "haploid" outfit of chromosomes (in Drosophila, 4; in the frog, Rana fusca, 12; in the guineapig, 8; in the rabbit, 22); and that in the fertilised ovum, resulting from the union of two gametes, there is a double or "diploid" outfit of chromosomes (in Drosophila, 8; in the frog, 24; in the guineapig, 16; in the rabbit, 44). The individual chromosomes often have distinctive forms, so that in the diploid cells it is easy to recognise the paired chromosomes which belong to one another (cf. Fig. 16).

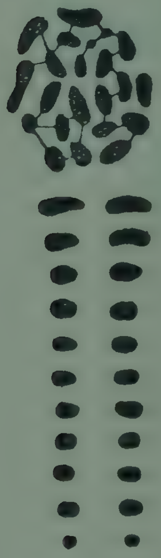

Fig. 16.

Chromosomes of diploid cells of the female of a bug, Anasa, shown above in their natural arrangement, and below ranged serially in pairs. (After Wilson.)

At some particular moment in the lifetime of the parent organism, the diploid cells, by a "reduction division", give rise to cells each of which has only a haploid equipment of chromosomes, the paternal and the maternal chromosomes being separately distributed. The cells thus arising may, as in all the higher animals, directly function as gametes; or they may (as happens, above all, in the case of most plants), after a variable number of divisions which do not affect the chromosome equipment, ultimately produce the gametes. This chromosome distribution in the reduction division could very well form the basis of the whole process of mendelian separation. I will explain this briefly by a systematised example.

Let us suppose that we have a living organism of which two stocks exist, and that each of these has a haploid set of three chromosomes, one of them having the chromosomes *A*, *B*, and *C*, and the other the chromosomes *a*, *b*, and *c*. Let us further assume that these chromosome distinctions *A-a*, *B-b*, *C-c*, are the causes of three independent mendelising fundamental differences between these two stocks.

All the diploid cells of a hybrid formed from the two stocks would then have the diploid chromosome outfit *Aa*, *Bb*, *Cc*. When the reduction division occurs in such a hybrid, we shall once more get cells each of which will have a haploid chromosome set. It is equally probable that this set will consist exclusively of paternal chromosomes, or exclusively of maternal chromosomes, or of any one of the possible minglings. Actually possible are the eight following haploid chromosome equipments *ABC*, *ABc*, *AbC*, *Abc*, *aBC*, *aBc*, *abC*, *abc*; and we should expect that, taking a sufficiently large number of cells, every eighth of them would contain one of these kinds of chromosome equipments. In other words, we should expect that such a hybrid would produce eight different kinds of gametes with equal frequency. This train of reasoning leads us to suppose that in actual fact the eight kinds of gametes must be produced which, according to the mendelian theory, a hybrid having the formula *Aa*, *Bb*, *Cc*, does in fact produce.

We might also very well suppose that the distribution of the heredity factors to the gametes of a hybrid has its physical foundation in these cytological processes.

Now, according to the latest investigations, this really does seem to be the case, though with a very important reservation.

The mere fact that in the case of all the organisms which have as yet been carefully studied there are many more mendelising heredity factors than there are pairs of chromosomes, suffices to show that the foregoing hypothesis cannot completely account for the phenomena.

The individual chromosomes are themselves made up of parts, which we will call chromomeres.[1] It is likely that before the reduction division there already goes on between the chromosomes of a pair a process of exchange of these elements; that is, an exchange of more or less lengthy portions of the chain of chromomeres. We can best picture this to ourselves as a tearing across of the two chromosomes at some point in their length, and a mutual exchange of the two resulting fragments. In this way there will necessarily come into existence chromosomes composed of both paternal and maternal elements (see Fig. 17).

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[1] The term "chromomere", as used in the present work, is not perfectly

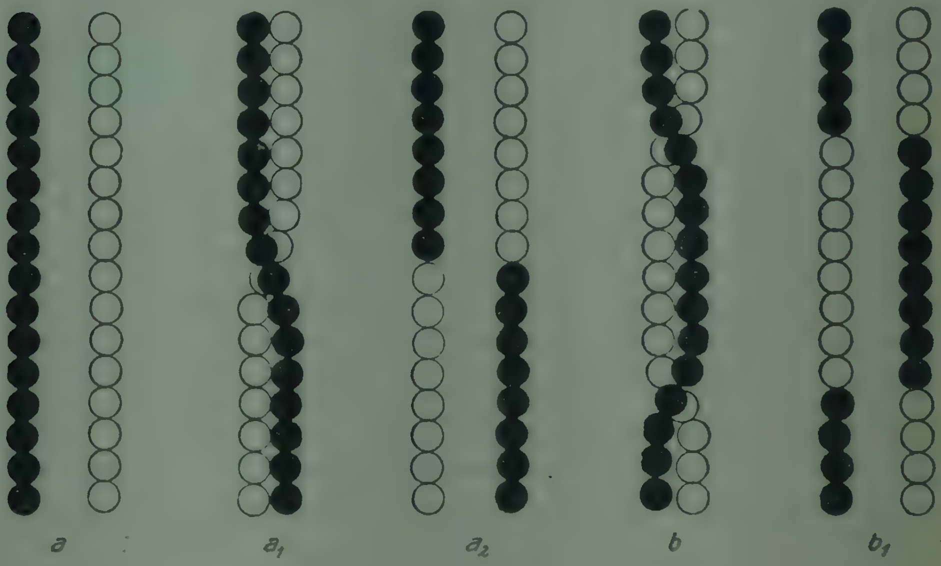

FIG. 17.

Diagram to illustrate the exchange of chromomeres between two homologous chromosomes. a, a_1, a_2 show the origination of mixed chromosomes (a_2) by a tearing across of the chromosomes at one spot ("single crossing over" of the American authors). b, b_1 show the origination of mixed chromosomes by a double exchange ("double crossing over" of the American authors).

It is extremely probable that the idioplasmatic basis of a racial difference which mendelises as a unity must be sought

identical with what British and American mendelians term "gene". When the elements of a chromosome are being considered as morphological units, i.e. structurally, the authors speak of them as chromomeres. When the authors are considering the same elements genetically, i.e. functionally, they term them "Erbeinheiten". This latter term, in the sequel, we shall usually translate "gene". "Erbfactor" having a somewhat wider significance than gene, will in most instances be translated "heredity factor".—TRANSLATORS' NOTE.

in a difference in the structure or in the chemistry of two homologous chromomeres.

If, when the gametes are being formed in a heterozygotic hybrid containing *n* factors, as many varieties of reproductive cells are produced as there are possible combinations among the *n* factors, we may, in accordance with the foregoing way of considering the matter, suppose the cause to be that: first of all, in the reduction division, the two chromosome sets undergo a perfectly free distribution, as described above on page 73, so that there arise all possible combinations of paternal and maternal chromosomes; and, secondly, that there occurs in addition an exchange of parts of two homologous chromosomes. Thus there is a twofold mechanism to secure the distribution of both kinds of inheritance, to bring about the formation of all possible combinations. But whereas the individual chromosomes of a chromosome set are in general independent of one another, so that in the reduction division they are distributed among the daughter-cells freely and quite independently of one another, the individual chromomeres of a chromosome are more or less firmly interconnected. We are not to suppose that a chromosome disintegrates into all of its chromomeres, and that these are then freely interchanged with the likewise disintegrated chromomeres of the homologous chromosome; but that when chromomeres are interchanged it is in groups or series.

The result of this must be that two mendelising heredity factors contained in two different chromosomes will mendelise freely and independently of one another, that is to say that the four gametes representing the four possible combinations will be formed in equal numbers; whereas two mendelising heredity factors which depend upon differences in the structure of two chromomere pairs of the same chromosome will not mendelise freely, but of the four possible gametes those will be formed in greater numbers in which the two heredity factors are associated in the combination in which they have entered into the crossing. In other words, *two heredity factors which are situated in the same chromosome must exhibit the phenomenon of the coupling of factors described above on page* 69.

The exchange of chromomeres would itself suffice to explain free mendelian separation and the phenomena of coupling. Even if nothing more were to take place than such an exchange of chromomeres in the absence of an exchange of entire chromosomes, the heredity factors localised in two different chromosomes would mendelise freely, whereas the heredity factors localised in one and the same chromosome would exhibit couplings. But there are numerous cytological observations leading us to infer that in addition to the exchange of chromomeres which must certainly occur in the opening phases of nuclear division, there ensues at a later stage an exchange of entire chromosomes—the result being that, as previously said, there is a twofold mechanism to bring about the distribution of the individual differences.

From this purely theoretical conception we can draw further inferences which are fully confirmed by experiments upon hereditary transmission, thus putting the whole theory of the cytological basis of mendelian separation upon a still firmer foundation. The first inference is that, if the theory is correct, every organism must have as many groups of more or less closely coupled heredity factors as it has haploid chromosomes. This is actually known to be the case in Drosophila, the fruit-fly, which is the one organism as yet subjected to thoroughgoing investigation from this standpoint. In Drosophila we know (thanks to the pioneer labours of Morgan and his school) that there are more than one hundred mendelising heredity factors, and these form four groups of factors which constantly exhibit the coupling of factors. Drosophila, however, as we have already learned, actually has four chromosomes.

The second inference is as follows. If the chromomeres of a chromosome form a chain, and if before the reduction division whole sections of the chain are interchanged between two homologous chromosomes, then two chromomeres situated at the opposite ends of a chromosome will be completely separated from one another by any such crossing over of the chromosomes, and will subsequently be distributed to two different daughter-cells, whereas the nearer two chromomeres lie to one another within a chromosome, the smaller is the probability that the laceration of the chromosome will occur between them. This means that there must be degrees in the intensity of coupling;

that some heredity factors of a "linked group" must be strongly coupled and others weakly coupled—but that, for a given pair of coupled heredity factors, the strength of the coupling must always remain identical. This inference is likewise substantiated by experiments on such organisms as have hitherto been sufficiently studied. In Drosophila, we can actually draw diagrams of the individual chromosomes showing how the heredity factors must be placed in them. Let me illustrate this by example. A hybrid $Aa\ Bb$ which is heterozygotic in two factors A and B, forms, if there be no coupling, that is to say if the two factors are situated in different chromosomes, the four gametes AB, Ab, aB, ab, in equal numbers. If, on the other hand, there is coupling, if the heredity factors are present in the same chromosome, then the gametes will be formed in the ratio $n : 1 : 1 : n$, or $1 : n : n : 1$, according as the hybrid has been formed by crossing AB with ab or by crossing Ab with aB.

The ratio $1 : n$ varies with the strength of the coupling, but is always less than 1. To facilitate comparison, I shall henceforward represent this ratio $1 : n$ as a decimal fraction.

In Drosophila the factors forming a series which I shall denote Y, W, V, M and R, and whose effects are shown in the following table, are coupled one with another to a varying degree of intensity.

Factor	*Effect*
Y	is essential to the formation of the dark pigment of the body. All yy fruit-flies have a yellow tint of body.
W	is essential to the formation of dark pigment in the eyes. All ww fruit-flies have white eyes.
V	is likewise essential to the formation of dark pigment in the eyes. All vv fruit-flies have vermilion eyes.
M	is essential to the proper growth of the wings. All mm fruit-flies have short "miniature" wings.
R	is likewise essential to the proper growth of the wings. All rr fruit-flies have atrophic, stumpy, "rudimentary" wings.

The varying strength of the coupling of these five factors, as worked out by Morgan in his experiments, is shown in the following table:

1	2	3
Coupled factors	Results obtained for 1 : *n* by actual experiment	The same value reduced to decimals
YW	354/32218	0.011
YV	2117/6221	0·340
YM	1054/3063	0·344
YR	605/1420	0·426
WV	4336/13395	0·324
WM	7591/22910	0·331
WR	894/2136	0·419
VM	50/1640	0·030
VR	183/850	0·215
MR	1562/9295	0·168

If we assume the ratio 1 : *n* (which has been worked out to express the intensity of the coupling of two factors) to be a measure of the distance between them in the chromosome, then we may infer that the distance from *Y* to *R* is greater than that between any other of the pairs, and that the other factors under consideration must be situated in relation to one another as shown in Fig. 18.

From this wholly theoretical way of regarding the matter it follows, of course, that if we have, on the ground of our theory, worked out for *Y-W* a coupling ratio and therefore a separation of 0·011, and for *W-M* a coupling ratio and therefore a separation of 0·331, then for *Y-M* there must be a coupling ratio and a separation of 0·011 + 0·331. Were there any inconsistency about the results of such calculations—if, that is to say, the reckoning of the position of a factor based upon a study of its various couplings did not always give us the same point in the imagined length of the chromosome—then the whole theory would be at fault. It is precisely because no such inconsistencies arise that the theory has to-day become so firmly established. A glance at the foregoing table shows that in actual fact the coupling ratio found for the factors

Y-M is 0·344, which is approximately = 0·331 + 0·011; and that, in general, the various results approximately tally. The only divergence from theory is that the coupling ratio for two factors as discovered by experiment (for instance, the ascertained figure 0·426 for *Y-R*) is always somewhat smaller than the figure calculated for the same two factors from their coupling with intermediate factors. Thus for *Y-R*, as an examination of Fig. 18 shows, the calculated figure is 0·011 + 0·324 + 0·030 + 0·168 = 0·533. But it is a legitimate inference from the theory that the directly ascertained coupling ratio should be somewhat smaller than the calculated ratio; and, indeed, the difference between the experimental and the calculated ratio must be greater in proportion as the coupling ratio is absolutely greater, that is to say the more widely separated two factors are in the chromosome. This results from the circumstance that, when between a pair of chromosomes a double exchange of portions is effected (b, Fig. 17) then the widely separated chromomeres become closely approximated in the same chromosome.

Conversely, from the extent of the difference between the experimental and the calculated coupling ratios, we can draw a conclusion as to the frequency with which such a double exchange occurs in the particular species.

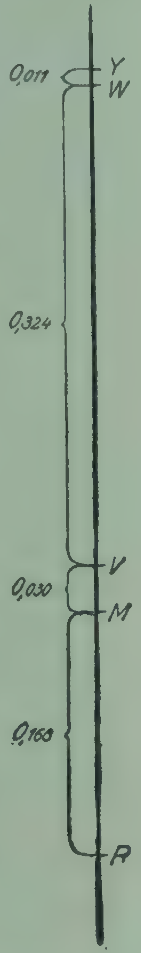

FIG. 18. Spatial distribution of a number of heredity factors in a chromosome of Drosophila.

The coupling of factors is not always, as in the foregoing instances, no more than partial. There may be complete coupling, inasmuch as two different factors may mendelise together invariably, never being free from one another. Such cases of the absolute coupling of several factors (usually termed "multiple allelomorphism" in the American and English literature on the subject) have been discovered in all organisms hitherto adequately studied. Apart, however, from the complete coupling with the sex factors, they all depend upon the circumstance that the factors in question are the outcome of several differences in the minute structure of one and the same pair of chromomeres—that is to say that they are "unilocal" factors.

In the case of all the organisms which have been closely studied we are acquainted with a number of series of such absolutely coupled factors, and these have been termed "allelomorph series" and "series of unilocal factors".

In the rabbit, for instance, such a series of unilocal factors determines the colour sequence white-sooty-chinchilla. An instance of the occurrence of unilocal factors has likewise been noticed in human beings.

c. Hereditary Transmission of Sex

The elaborate experiments of Correns had already made it clear that in bisexual organisms the sex distinction itself is transmitted by inheritance in accordance with the laws of separation. In this process, one of the sexes always behaves as a homozygote, is "homogametic", whilst the other behaves as a heterozygote, is "heterogametic".

In the fruit-fly, for example, the sex distinction is determined by a "heredity factor" which we may call X. All females are XX and all males are Xx. Thus every act of copulation is a recrossing in accordance with the following scheme:

$$\begin{array}{lr} XX \times Xx & P_1 \\ 50\%\ XX,\ \ 50\%\ Xx & F_1 \end{array}$$

This simple assumption suffices to account for the fact that, in the course of reproduction, males and females appear in approximately equal numbers.

Although we have here denoted the sex distinction by the customary mendelian formula, this does not mean that it is due to just such a "heredity factor" as the heredity factor E which determines the difference between a zygomorphic and a radial Antirrhinum stock. Experiment has, indeed, shown that a whole undivided chromosome is determinative of the difference of sex. In the fruit-fly, the body cells, as already said, have normally four pairs of chromosomes. Of these four pairs of chromosomes, three, both in the male and in the female, consist of two identical partners; but the fourth pair of chromosomes consists of two identical partners in the female only, whereas in the male the two partners are different, as is plainly shown in Fig. 19. We say that the female has two *XX* chromosomes, and that the male has an *X* and a *Y* chromosome.

In the female, during the maturation of the ovum (see the left lower part of Fig. 19), there is produced only one kind of ova, each of which contains only one of each of the paired chromosomes, and has therefore only one *X* chromosome.

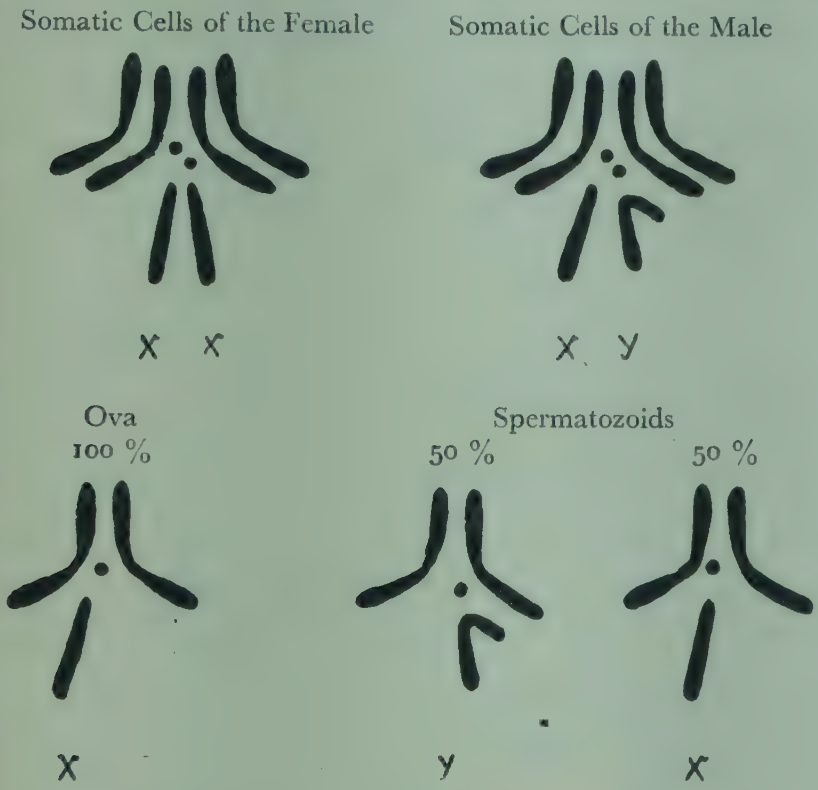

FIG. 19.
Chromosome sets in males and females of Drosophila.

In spermatogenesis, on the other hand (see the right lower part of Fig. 19), there originate two kinds of spermatozoids: one kind containing an equipment of single chromosomes including *X*, and the other half containing an equipment of single chromosomes including *Y*. The outcome of this is that when ova are fertilised two sorts of insects are produced, differing according to the kind of spermatozoid with which the particular ovum has been fertilised. Thus we have, on the one hand, insects containing two *X* chromosomes, and these are females; and, on the other hand, insects containing one *X* and one *Y* chromosome, and these are males.

In the case of many other organisms, likewise, both animals and plants, it has been shown that a particular chromosome is determinative in the production of sex. The remarkable thing is that sometimes one of the sexes is homogametic, and sometimes the other. In Drosophila, in all the plants hitherto studied, and in all mammals, including our human kind, the males are *XY* and the females *XX*. Conversely, in all butterflies and birds hitherto studied, the females are *XY* and the males *XX*.[1] Thus in the case of butterflies and birds, only one kind of spermatozoids is formed, all of them being *X*, whereas two kinds of ova are formed, half of them being *X* and half of them *Y*.

In the fruit-fly, and in a great many other organisms, the *X* and the *Y* chromosomes are clearly distinguishable in shape, the *Y* chromosomes being sharply bent into a hook, and the *X* chromosomes being straight. In certain other organisms, the *Y* chromosomes are considerably smaller than the *X* chromosomes; and in yet other organisms the *Y* chromosomes are lacking. In these last, therefore, the homogametic sex has n chromosomes, whereas the heterogametic sex has $n - 1$. Such is the case, for instance, in the bug Anasa tristis. Here (see Fig. 16) the males have 21 chromosomes in the body cells and the females have 22. Of the spermatozoids, half have 10 chromosomes, and the other half 11; but all the ova have 11 chromosomes. Consequently the union of an ovum with a spermatozoid containing 10 chromosomes produces an animal with 21 chromosomes, that is to say a male, whereas the union of an ovum with a spermatozoid containing 11 chromosomes gives an animal containing 22 chromosomes, that is to say a female.

Not in all organisms, however, are the sex chromosomes readily recognisable as such, for the *X* and the *Y* chromosomes may morphologically (that is to say, to outward appearance) be extremely similar or even identical. Probably this is the case in the human species. Still, the perfectly unambiguous results of investigation in those plants and animals in which the sex chromosomes are distinguishable, have established beyond all reasonable doubt that, as a general biological rule, the distinction between males and females is determined by a particular pair of chromosomes, namely the sex chromosomes.

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[1] Of course, if we prefer to do so, we may say that the females are *XY*, and the males *YY*.

Now, the sex chromosomes have one important peculiarity. In most organisms, the *Y* chromosomes are wholly ineffective, by which I mean that such "heredity factors" as may be present in them, do not manifest themselves in any way.

This has very important consequences for the hereditary transmission of qualities which are influenced by heredity factors contained in the sex chromosomes. It accounts for a phenomenon which is at first sight difficult to understand, that of "sex-linked inheritance", which plays an especially large part among human beings. We shall best gain a knowledge of the laws of sex-linked inheritance by the study of an obvious example in Drosophila. In that insect, the factor *W* which renders possible the formation of the dark colour of the eyes (all *ww* fruit-flies have white eyes) is contained in the sex chromosome.

If we cross a normal red-eyed female fruit-fly with a white-eyed male, the F_1 generation consists of red-eyed males and red-eyed females. The F_2 generation, bred from these F_1 insects, consist of red-eyed females and of both red-eyed and white-eyed males.

Approximately half of the males are red-eyed and the other half white-eyed. Thus the white-eyed grandfather has transmitted the characteristic colour of his eyes to half of his grandsons, but to none of his granddaughters. This method of hereditary transmission will become readily comprehensible from a study of the subjoined diagrammatic genealogical tree (Fig. 20, p. 84).

Thus the peculiar mode of the hereditary transmission of the eye-colour is readily explicable from the coupling.

In F_2 of this crossing, no white-eyed females appear, but it is possible to obtain white-eyed females in a different way. If we cross with white-eyed males a number of the dark-eyed females obtained in F_2 of the crossing just described, the

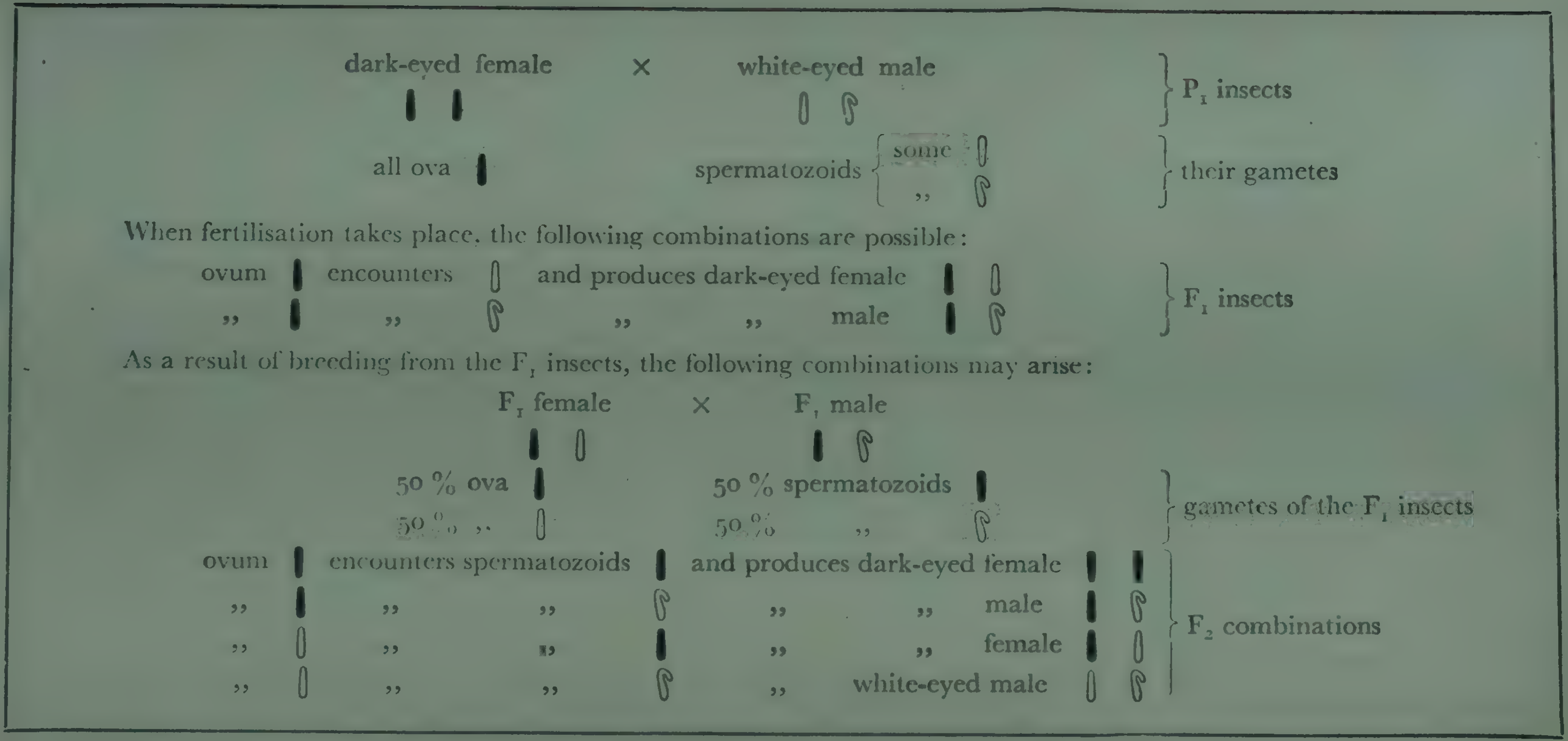

dark-eyed female × white-eyed male
▮ ▮ ▯ ʃ
P₁ insects
all ova ▮
spermatozoids some ▯
,, ʃ
their gametes
When fertilisation takes place, the following combinations are possible:
ovum ▮ encounters ▯ and produces dark-eyed female ▮ ▯
,, ▮ ,, ʃ ,, ,, male ▮ ʃ
F₁ insects
As a result of breeding from the F₁ insects, the following combinations may arise:
F₁ female × F₁ male
▮ ▯ ▮ ʃ
50 % ova ▮
50 % ,, ▯
50 % spermatozoids ▮
50 % ,, ʃ
gametes of the F₁ insects
ovum ▮ encounters spermatozoids ▮ and produces dark-eyed female ▮ ▮
,, ▮ ,, ,, ʃ ,, ,, male ▮ ʃ
,, ▯ ,, ,, ▮ ,, ,, female ▮ ▯
,, ▯ ,, ,, ʃ ,, white-eyed male ▯ ʃ
F₂ combinations

FIG. 20.

Diagram of an instance of sex-linked hereditary transmission. ▮ denotes an *X* chromosome containing the factor *W*. ▯ denotes an *X* chromosome with *w*. ʆ (does not appear in this figure) denotes a *Y* chromosome containing the factor *W*. ʃ denotes a *Y* chromosome with *w*. The reader will please remember that *W* contained in a *Y* chromosome is inoperative.

two sexes that result will consist of dark-eyed and white-eyed females and of dark-eyed and white-eyed males in equal numbers. That arises in the following way. Half of the F_2 females produced in the first experiment have the chromosome set ▮ ▯, and when these insects are paired with ▯ ʃ males we get the four combinations:

▮ encounters ▯ and produces dark-eyed females,
▮ ,, ʃ ,, ,, ,, ,, males,
▯ ,, ▯ ,, ,, white-eyed females,
▯ ,, ʃ ,, ,, ,, ,, males.

Thus theoretical considerations show that males of both kinds and females of both kinds will be formed in equal numbers, as actually occurs in the experiment.

The obverse crossing to that of our first experiment (the one diagrammatised in Fig. 20), a crossing between white-eyed females and dark-eyed males, gives in F_1 only dark-eyed females and white-eyed males. In F_2, females of both kinds and males of both kinds appear with equal frequency. This is shown in the diagram on page 86 (Fig. 21).

In human beings the hereditary transmission of many sex-linked characters occurs in precisely the same way. Let us diagrammatise this for the transmission of red-green blindness. Here the disturbance of the colour sense depends upon a recessive factor *r* which is contained in the sex chromosome. All *rr* persons are red-green-blind.

The possible crossings are shown in the diagram on page 87, Fig. 22.

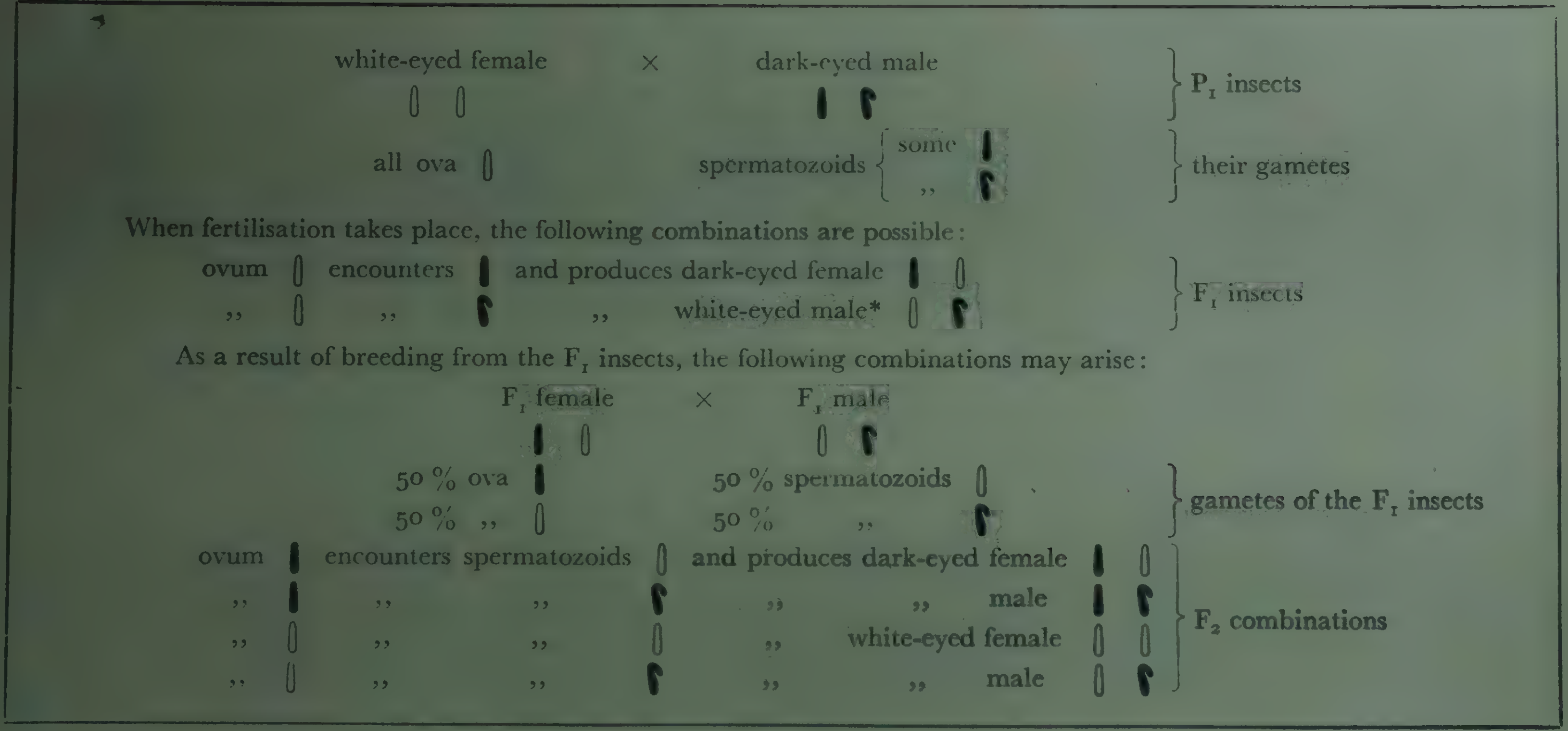
If we arrange the foregoing in a genealogical tree, we get:

normal-sighted woman × red-green-blind man

normal-sighted sons
normal-sighted daughters
a normal-sighted man × one of these daughters

half-sons normal-sighted
half-sons red-green-blind
all daughters normal-sighted

Here the morbid character in the man who marries a homozygotically healthy woman is not manifest in the children


white-eyed female
×
dark-eyed male
P₁ insects
all ova
spermatozoids
some
,,
their gametes

When fertilisation takes place, the following combinations are possible:

ovum encounters and produces dark-eyed female
,, ,, ,, white-eyed male*
F₁ insects

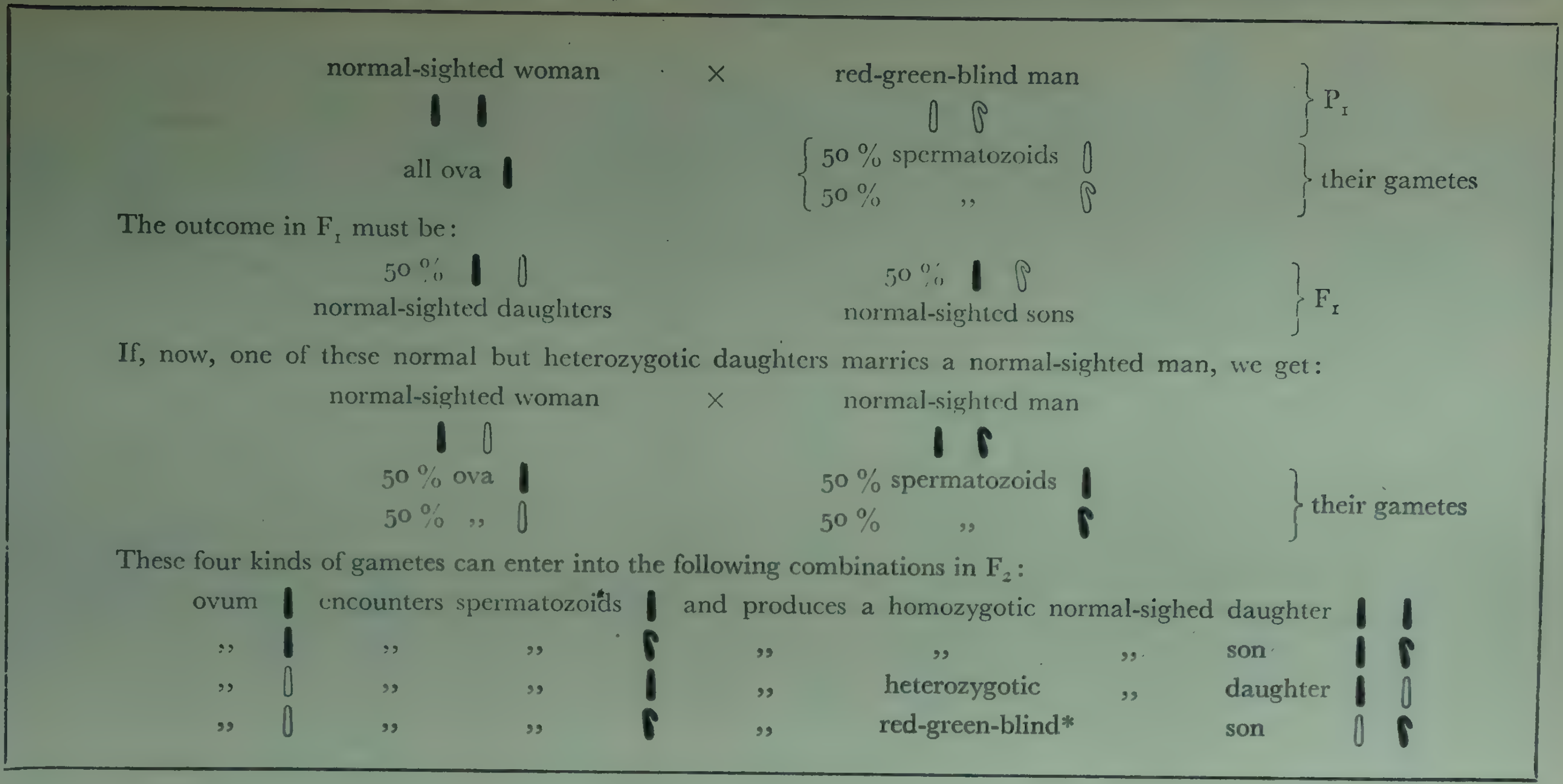
As a result of breeding from the F₁ insects, the following combinations may arise:

F₁ female
×
F₁ male
50 % ova
50 % ,,
50 % spermatozoids
50 % ,,
gametes of the F₁ insects
ovum encounters spermatozoids and produces dark-eyed female
,, ,, ,, ,, ,, male
,, ,, ,, ,, white-eyed female
,, ,, ,, ,, ,, male
F₂ combinations

FIG. 21.

Sex-linked hereditary transmission, this crossing being the obverse of the one diagrammatised in Fig. 20. For the explanation of the signs see description of that figure.

* *W* in *Y* chromosomes is inoperative.


normal-sighted woman × red-green-blind man
P₁
all ova
50 % spermatozoids
50 % ,,
their gametes
The outcome in F₁ must be:
50 %
normal-sighted daughters
50 %
normal-sighted sons
F₁
If, now, one of these normal but heterozygotic daughters marries a normal-sighted man, we get:
normal-sighted woman × normal-sighted man
50 % ova
50 % ,,
50 % spermatozoids
50 % ,,
their gametes
These four kinds of gametes can enter into the following combinations in F₂:
ovum encounters spermatozoids and produces a homozygotic normal-sighed daughter
,, ,, ,, ,, ,, ,, son
,, ,, ,, ,, heterozygotic ,, daughter
,, ,, ,, ,, red-green-blind* son

Fig, 22.

Explanation in the text.

* Because *R* in the *Y* chromosome is ineffective.

of the first generation, all of them being to outward appearance free from the taint; but the daughters of this marriage who enter into unions with healthy men transmit the infirmity to half of their sons. Or we can formulate the matter in another way as follows. Ostensibly healthy daughters of a man with a sex-linked hereditarily transmissible morbid character when fertilised by perfectly healthy husbands have sons 50 % of whom will manifest the same infirmity as their paternal grandfather. Of their daughters (the granddaughters of the affected male ancestor) 50 % will likewise transmit the infirmity to 50 % of their sons; and so on.

In our own species it has been possible again and again to trace such a morbid inheritance through several generations.

If the outwardly normal daughter of a red-green-blind father marries a red-green-blind man, some of her daughters as well as some of her sons will be red-green-blind. This is diagrammatised in the following figure:

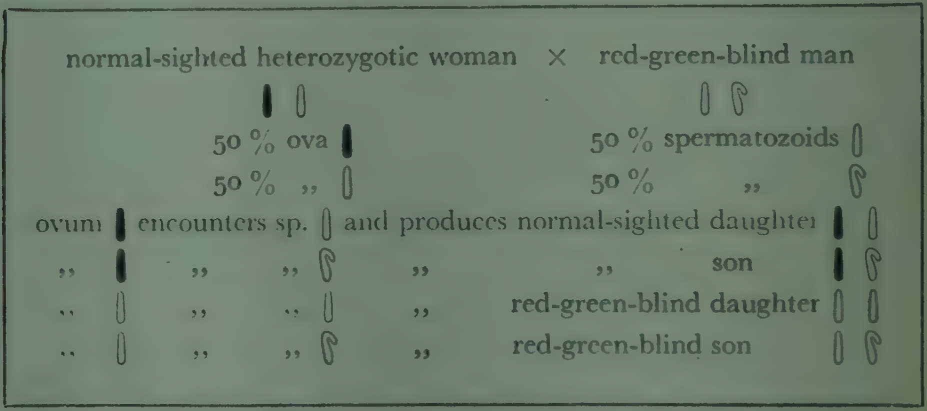

normal-sighted heterozygotic woman × red-green-blind man
50 % ova
50 % ,,
50 % spermatozoids
50 % ,,
ovum encounters sp. and produces normal-sighted daughter
,, ,, ,, ,, ,, son
,, ,, ,, ,, red-green-blind daughter
,, ,, ,, ,, red-green-blind son

FIG. 23.

If one of these red-green-blind daughters marries a normal-sighted man, we get:

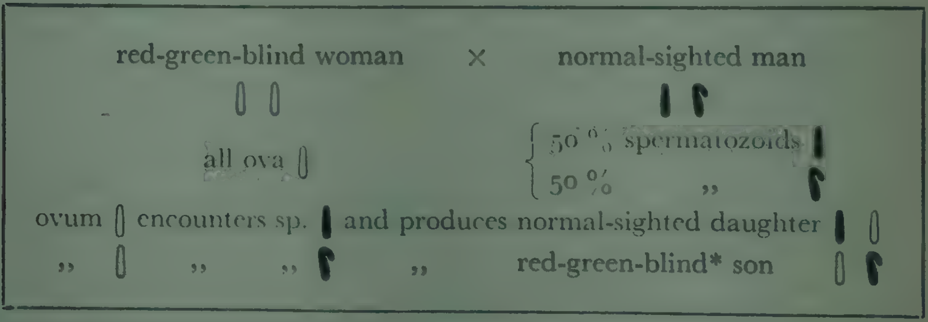

red-green-blind woman × normal-sighted man
all ova
50 % spermatozoids
50 % ,,
ovum encounters sp. and produces normal-sighted daughter
,, ,, ,, ,, red-green-blind* son

FIG. 24.

* Because *R* in the *Y* chromosome is ineffective.

Here the mother transmits the infirmity to all her sons, whereas her daughters are normal-sighted (but all of them heterozygotic!).

Further instances of this kind of inheritance in human beings will be described in a later section, that by Lenz on Hereditary Morbid Predispositions. It must be pointed out that observers were familiar with cases of sex-linked hereditary transmission in human beings long before this peculiar process of inheritance was rendered comprehensible by experiments on animals.

Sex distinctions have likewise a powerful influence on the exchange of factors, or crossing over (see above page 74). In many organisms, as for instance in Drosophila, crossing over occurs only in the female sex, that is to say during the formation of the ova, whereas in the male sex, during the formation of the spermatozoids, it is completely lacking, so that here all the factors localised in the same chromosome display an absolute coupling one with another. This is especially plain in the case described above on page 69. There we had to do with a crossing between a black normal-winged stock, *bb VV*, and a wild-coloured stumpy-winged stock, *BB vv*. The two factors *B* and *V* are situated in the same chromosome and consequently exhibit coupling. If we recross a hybrid female from this crossing with a doubly recessive male, *bb vv*, we learn from the composition of the offspring that such a female forms four kinds of ova, *BV*, *Bv*, *bV*, *bv*, in the ratio 1 : 5 : 5 : 1. Altogether different is what happens in the male. If we cross an F_1 male with a doubly recessive female, we get only two types in the offspring, only wild-coloured stumpy-winged and black normal-winged. It follows from this that the F_1 males have not formed four kinds of gametes, *BV*, *Bv*, *bV*, *bv*, but only two kinds of gametes, *Bv* and *bV*. The gametes *BV* and *bv*, which can only be formed by crossing over, are entirely lacking, which means that no crossing over occurs in the males.

This does not, however, apply to all organisms. In the silkworm moth, for instance, crossing over occurs only in the males, not in the females. This was, indeed, to be expected, since in the silkworm the males are homogametic, whereas the females are heterogametic. In rats, again, we know that crossing over occurs in both sexes, but in the males less often than in the females. What happens in this respect in human beings is not yet known.

Inheritance to members of the same sex as the transmitting parent may exhibit other complications besides those due to the coupling of many heredity factors to the sex factor. For instance, the dominance of a quality may differ in the two sexes: or a heredity factor may manifest itself only in one sex while remaining inoperative in the other; with the result that, for example, two races of females are distinguishable; whereas the males belonging to the respective races are not outwardly distinguishable, and nevertheless inherit the racial characteristics of the females of their race. But it would lead us too far afield to describe all such instances here, and the reader must be referred to the specialist literature of the topic.

Inasmuch as sex is transmitted by inheritance in accordance with the laws of separation, sex must be determined for every individual at the moment of fertilisation. In human beings, all the ova have the same sexual predisposition; but as regards the spermatozoids, half of these transmit the predisposition for masculine characters, and the other half the predisposition for feminine characters. We should therefore expect the numerical ratio between the sexes to be 1 : 1. It is, however, well known that the ratio of female births to male births is not 1 : 1. In Germany, for instance, 100 girls are born to 105·2 boys. These are the figures for the children born alive, but if we take into account all stillbirths and all abortions (insofar as in the latter the embryo is sufficiently developed for the sex to be determinable), we get a ratio of 100 females to nearly 150 males. The cause of this excess of male births is still unknown. It may be that, of the two kinds of spermatozoids which every man produces, those with the masculine equipment are better fitted to traverse the long distance from the vagina to the Fallopian tube, so that a kind of selection occurs in this matter. An exhaustive experimental study of this question would have great practical value apart from its theoretical interest, for, from all we know to-day concerning the hereditary transmission of sex, it would appear that here we are concerned with the only possibility of deliberately determining sex in human beings—with the only way in which there is some reason to expect that we may learn how to influence the sex of a child about to be procreated.

The researches of A. Bluhm have shown that in mice the pairing of chronically alcoholised males with non-alcoholised females produces many more males than when the fathers are not alcoholised. It seems probable, therefore, that the two kinds of spermatozoids have a different susceptibility to alcohol.

It is very improbable that in human beings there can be any way of changing the sex of an embryo after fertilisation has occurred. It is, indeed, likely enough that by the introduction of the hormones of the adult reproductive glands into the circulation of very young embryos in which sexual differentiation has not yet become manifest, a certain degree of modification of the sex characters may be brought about; but we can hardly expect to produce in this way anything other than more or less developed hermaphrodites, such as the calves known as "freemartins".[1] Concerning these matters, however, I must once more refer the reader to the specialist literature of the topics, a further consideration of which would lead us too far from our present subject.

There are, however, organisms, frogs for instance, in which a complete transformation of one sex into the other is possible.

A different problem is, how far in the higher mammals and in human beings it is possible at a comparative late stage of development to modify the "secondary" sexual characters and the reproductive instinct. Universally known are the effects of castration and of spaying. Just as in other vertebrates the early removal of the reproductive glands brings about a marked change in bodily structure and in temperament—the bull becoming an ox; the stallion, a gelding; the boar, a porker; the rooster, a capon; the hen, a poularde—so in man, likewise, castration leads to the production of the eunuch type and character. If after the removal of the reproductive glands, a gland of the opposite sex be ingrafted, if, for instance, we remove the testicles of a guineapig and implant an ovary in the animal's abdominal cavity, under the influence of the new kind of sexual hormones which are now circulating in the organism subjected to the experiment the bodily structure and .the reproductive instincts of the creature are plainly modified. Such "feminised" male guineapigs develop lacteal glands so that they can give suck to young guineapigs, and they show in various other respects obviously feminine qualities.

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[1] When a cow has twin calves, the embryonic calves have a common placental circulation, so that their respective hormones are readily interchanged. If the twins are of opposite sex, the female calf, spoken of as a freemartin, is found to have undergone certain modifications in the direction of maleness, and is always sterile. Apparently the reproductive hormones of the male embryo are formed earlier and are more potent than those of the female, and give the development of the female a twist.—TRANSLATORS' NOTE.

d. Mendelian and non-Mendelian Heredity

The partial coupling and still more the absolute coupling of factors show that the mendelian laws of separation do not apply without reservations to all racial differences. It is obvious that only those racial differences are transmitted in accordance with the laws of separation, which are mechanically determined by slight differences in the structure of homologous chromomeres. Moreover, a mendelian separation occurs only when the whole complicated apparatus of the exchange of chromomeres, of the distribution of chromosomes, etc.—that is to say the entire mechanism of the reduction division—operates undisturbed. But as soon as we proceed to cross races characterised by such extensive difference in nuclear structure that the working of this mechanism is disturbed in one way or another, we find that the mendelian process of assortment is likewise disturbed. Consequently, while greatly differing species can often still be crossed, and can even produce fertile hybrids, the subsequent hereditary transmission of characters will occur in ways differing markedly from one individual case to another.

We find, indeed, often enough, that closely allied species which are none the less very different morphologically, will when crossed continue to mendelise according to rule, and will in the F_2 generation give rise to an incredible multiplicity of new combinations—but the majority of hybrids between different species behave in another way than this.

Here, however, we need not attempt to discuss the whole problem of the hybridisation of species, seeing that when different races of man are crossed the hereditary transmission of their racial characters occurs (as far as our present knowledge goes) mainly in accordance with the laws of mendelian separation. Nothing is known as to crossings between members of the human species and those of other species of primates, the anthropoid apes. Experiments of the kind could, of course, be made by way of artificial insemination. It is most unlikely that such an attempt at pairing would produce a viable embryo; but it would be a pity if no such experiments were to be tried before the anthropoid apes become extinct.

In view of the theoretical considerations here adduced concerning the cytological cause of mendelian separation, we have likewise reason to expect that hereditary transmission in accordance with other laws than the mendelian will occur in the case of all racial differences which are based, not upon peculiarities of the chromosomes, but on those of some other part of the idioplasm. Differences in the structure of the remainder of the thread equipment of the cell nucleus, in the structure of the centrosomes, etc., must certainly produce differences of one kind or another in the finished organism. Now, in the case of many plants we know of certain racial differences which definitely do not mendelise, even when all the other differences of the races concerned are subject to the laws of separation. We have, moreover, some ground for supposing that certain among such non-mendelising differences must be determined by the structure of the chromatophores (colour-carriers). Very numerous observations on plants combine to show that hereditary differences in their vitality are transmitted in accordance with other laws than the mendelian, that is to say that the ultimate mechanical developmental cause of the differences in question must be sought elsewhere than in the cell nucleus. Speaking generally, however, we know as yet so little about this matter that its discussion here would be out of place—all the more seeing that we know nothing at all about unmistakably non-mendelian racial distinctions in the human species. It will be enough for the reader to bear in mind that the mendelian laws of separation do not prevail universally, and that, sooner or later, in the case of man no less than in that of other organisms, we are likely to encounter phenomena of hereditary transmission which do not occur in accordance with these laws.

e. SUMMARY

Let me now summarise what we have learned about combinations. A second main cause for the fact that children differ from their parents and from one another consists in this, that in sexual reproduction the union that is effected is usually between two different idioplasms, two different hereditary trends, or however you like to phrase it; and that in the gametes of the hybrids these different hereditary trends are distributed among the individual cells in accordance with very complicated laws and rules. In the offspring of two individuals that are crossed there arise all the possible new combinations of the original differences.

For the most part, the new combinations of racial differences occur according to the laws discovered by Gregor Mendel—laws which have been considerably amplified and perfected during recent years. There are, however, certain racial differences, and a great many differences between species, which are transmitted by inheritance, and which combine one with another, in accordance with other rules than the mendelian, rules which are as yet very little known.

The perpetual kaleidoscopic origination and disappearance of new combinations of a certain number of primary racial differences is the chief cause of the inheritable variations that occur in organisms which reproduce sexually. This applies to man as well as to other organisms.

3. MUTATION (IDIOVARIATION)

This ringing of the changes in the way of the combination of a certain number of fundamental differences is, as we have just learned, a means for the production of an enormous number of variations, and some biologists seriously contend that all hereditarily transmissible variations are, essentially, combinations. But this view is certainly erroneous. There are whole classes of organisms, such as the bacteria, the blue-green algae (cyanophyceae), many other algae, and quite a number of filamentous fungi (hyphomycetes), whose reproduction is exclusively asexual; and nevertheless in them, too, hereditarily distinct new races arise. But, altogether apart from this, there can to-day be no doubt whatever that, even in the organisms which reproduce sexually, new hereditary distinctions are continually arising before our very eyes.

When in any particular instance an individual comes under our notice differing from its parents and from its brothers and sisters, it is only by prolonged investigation during perhaps several decades, and by the analysis of genealogical trees for a number of generations, that we can determine whether we have to do with nothing more than a new and rare combination resulting perhaps from coupling or from other causes, or may be from an irregular separation, or whether we have to do with a previously non-existent fundamental distinction. If, to-day, erroneous ideas concerning mutations are so widely diffused, this is because so small a number of organisms have as yet been adequately studied.

A critical scrutiny of all the material hitherto available gives some such picture as the following. In the case of every species of organisms hitherto carefully investigated, mutations occur from time to time, appearing with very irregular frequency, and dependent as a rule upon entirely unknown causes. The great majority of them are due to the origination of a new mendelising fundamental distinction. Generally when we cross the new kinship with the parent race, the characters of that parent race display a more or less marked dominance over the characters that have recently appeared. Still, we also come across a great number of genuine mutations in which the new character proves dominant when a crossing is effected with the parent race. Far more exceptional in our experience are mutations in which the new differential characters do not mendelise, but are transmitted in accordance with some other laws than the mendelian. This observation harmonises with what we have already learned, namely that the great majority of all race distinctions do mendelise, and that the hereditary transmission of racial differences in accordance with other laws than the mendelian is rare.

No precise data can as yet be given concerning the frequency of mutations. Unless a highly specialised method of experimentation has been adopted, it will only be possible to note and to recognise a small proportion of the mutations that actually occur. We can say no more than this, that in the case of those organisms which have so far been studied with some thoroughness (there are only two, Drosophila and Antirrhinum), among one hundred offspring of a pair of parents there will always be found several individuals with mutations. It follows that mutations are far commoner than biologists have been wont to believe.

To-day mutations of this kind are termed "factor mutations", and it is assumed that they are brought about by a change in one of the chromomeres. Such a change may occur either in a diploid or in a haploid cell. If a factor mutation occurs in a diploid cell, then the two chromosome sets are independent of one another; this meaning that when a mutation occurs in one of them, the corresponding chromosome in the other set usually remains unaffected. Factor mutations are obviously very frequent in human beings. We may presume that the greater part of all the numerous hereditary errors of development are the outcome of such factor mutations.

A second kind of mutations depends upon changes in the chromosome equipment. A race in which one of the chromosome pairs has been doubled is, according to all that we have learned from the study of plants, manifestly different in one way or another from the normal race. In the thorn-apple (Datura stramonium), for instance, the appearance of many new racial types in experimental cultures is explicable from the fact that one chromosome pair, or two pairs, or many pairs, or all the pairs, have been doubled.

Mutations of a third kind form a sort of intermediate link between mutations and combinations. For example, in the genus Oenothera, through a crossing of the species, it is possible to originate on a large scale "constant" species, which, however, are able in a very high degree, probably owing to processes which remind us of the exchange of factors or crossing over, to give rise to new species.

We are to-day in a position to recognise other types of mutation more or less clearly sketched, but we have so little certain information about them that there is no occasion to describe them here in detail. As far as the human species is concerned, it is possible only in exceptional instances to recognise a mutation as such. Still, as already said, there is considerable evidence to show that the majority of mutations in human beings belong to the first of the before-mentioned three categories; that they are "factor mutations".

In most instances, factor mutations produce more or less pathological types, many of which have only a restricted viability, so that quite a number of them can exist only heterozygotically ("lethal factors"). The reason for this is almost self-evident. Any change blindly made in a complicated mechanism such as that of a repeater watch will almost inevitably interfere with its working, and can very rarely lead to the formation of a new harmony.

In the last analysis, mutation must underlie every formation of a new stock, for without it the development of fresh kinships and races, and still more of fresh species, is inconceivable. Here, however, we are mostly concerned with mutations which are not very conspicuous, with "minor mutations".

We know hardly anything about the causes of mutations. Most of those which have come under observation in thoroughly trustworthy experiments have been "spontaneous", though this of course only means that we do not know the cause. This much, indeed, we are entitled to say to-day, that mutations can probably be produced by very various influences, such as high temperatures, the effect of chemicals, of radium, of X-rays, etc.

As far as organisms reproducing themselves sexually are concerned, all the observations hitherto made incline to show that inbreeding, when of a marked degree, tends to make mutations more frequent. Nevertheless, we must be cautious in drawing this inference. Since the majority of mutations are recessive, and since inbreeding favours the mendelising-out of recessive types in a homozygotic form, when inbreeding is practised mutations are more easily discovered. This does not, however, mean that they may not occur just as frequently in other circumstances. Another possible exciting cause of mutations, is, perhaps, the crossing of markedly differing stocks, for then the whole mechanism of nuclear division and cell division, and above all that of the reduction division, is disturbed, and the two different sections of the nucleus are not properly adapted each to the other. But it is precisely in mutations belonging to this last category that it is apt to be so difficult to determine whether we have to do with a mutation or with an irregular separation. Besides, when we look into the matter closely we see (as was already pointed out on page 96) that in such cases there are almost insuperable theoretical difficulties in the way of drawing a sharp distinction between mutations and combinations. In this field the two categories of variations shade into one another.

It is very probable, first that it is a hereditary character of certain stocks to give rise to particular mutations with special frequency, and secondly that this predisposition is transmitted by inheritance in accordance with the laws of separation. We may picture the mechanism of such cases by supposing that certain chromomeres are exceptionally labile in structure, and that that is why mutations may occur with unusual facility.

Of great interest for human pathology are experiments which have been made to determine whether and to what extent a high degree of poisoning of a parent can affect the hereditary constitution of the offspring—can, that is to say, give rise to mutations. It is extremely difficult to draw trustworthy inferences regarding this matter from the data available in the case of human beings. If, for example, we find that the children of a man given to drink are of low grade in one way or another, we have to remember that as a rule the father will himself have become a drunkard only because he was already below par psychically speaking, and in that case nothing more may have happened than the transmission of his own hereditary equipment to his children. Or the children of a drinker may be of inferior quality because the hygienic and social conditions under which they have grown up have been much less satisfactory than those under which the children of healthy parents grow up. But when we turn to consider experiments on animals, these show that the chronic alcoholic poisoning of healthy animals does actually cause grave injury to the offspring, manifesting itself in the occurrence of an exceptionally large number of stillbirths and in the production of weakly and sometimes malformed young. Here we apparently have to do, not merely with an "after-effect" (see above page 37), but with a permanent injury to the idioplasm; for the second generation (the F_2 animals) of the alcoholised stock continue to show these defects, although only the P_1 animals were treated with alcohol, and not the F_1 animals. We have, then, to recognise that toxic and doubtless other harmful influences may affect the idioplasm and produce hereditarily transmissible injuries. We must, however, bear in mind that in such experiments on animals, the alcoholic poisoning is of a much higher degree than that to which even the most inveterate topers are exposed.

In such organisms as man, which reproduce sexually and breed promiscuously ("panmixia"), the three above-described categories of variation go on appearing continuously side by side and intermingled. It is as a rule very difficult—far more difficult than the layman is apt to suppose—to decide whether a difference between two human beings depends upon modification, combination, or mutation. Such a decision can only be arrived at, if at all, through very elaborate study. I think it essential to emphasise this fact at the close of the present chapter.

CHAPTER THREE

INFLUENCE OF VARIATION UPON THE COMPOSITION OF A PEOPLE. EFFECTS OF SELECTION

WHAT we to-day term a nation or a people is, biologically considered, a mishmash of the most extraordinarily diversified elements. If we were to take a couple of families of jackals from Asia Minor (Canis aureus), two or three more families of jackals from Northern Africa (Canis lupastar), and two or three families of wolves (Canis lupus), and were to allow them to interbreed promiscuously under the most favourable conditions possible, so that all the combinations might be preserved, and so that there should be no naturally selective elimination of various mutations such as bandiness of the legs, the appearance of the pug type, that of the greyhound type, exceptional length of the hair, etc.—then there would be produced a race of dogs which would be just such a composite admixture of strains as is a human nation or people to-day.[1]

It cannot be too emphatically insisted that that which is common to the people of one nation, such as the German, the British, or the French, and that which unites them as a nation, is not, properly speaking, their "race", but, first and foremost, a common speech and culture. Racial distinctions, such as the differences between the before-mentioned peoples, are never anything more than relative distinctions, insofar as the quantitative proportions out of which the mixture is made up in the various peoples differ to some extent, some racial elements being more strongly represented in one nation, and others in another. Moreover, unmistakably anthropological distinctions between nations by no means necessarily coincide with the boundary lines between folk-speeches.

If we find, as we do, that in a great nation numbering many millions of persons there are never to be found any two individuals exactly alike, but that there is always a marked "variability" in the individual characteristics, this is in part dependent upon the fact that the separate individuals have been modified to unequal extents (page 25), and in part upon the fact that from time to time mutations arise; but the difference between individuals depends mainly on this, that in such a mixed stock of human beings every act of reproduction leads to the formation of new combinations of mendelising and non-mendelising racial differences.

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[1] When making the comparison in the text, I have no intention to imply that the various racial constituents out of which a human people is composed were originally distinct "species" in the sense of the systematists.

Since modifications are not transmissible by inheritance, they cannot affect the hereditary composition of a people, no matter how great the influence of the method of nutrition, of the whole conduct of life, etc., upon the individual human being may be.

Mutations, on the other hand, and in certain circumstances (when selection is at work!), combinations, may greatly influence the hereditary composition of a people.

If some particular mutation (let us take the appearance of a hereditary predisposition to diabetes as an example) should appear only at rare intervals—say in a heterozygotic individual once in every ten thousand births—and if the diabetics were not eliminated by a vigorous process of selection, but were able to go on reproducing themselves as freely as the average population, then the percentage of diabetics in the population would continually increase, until sooner or later a time would come when almost the entire population would consist of diabetics. Thus interference with natural selection, taking the form of too far-reaching a hygienic and social protection of persons exhibiting mental or bodily mutations towards an inferior type, can lead to the degeneration of a racial stock unless care be taken to prevent the reproduction of the unfit, or at least to ensure that they shall not reproduce themselves in so effective a percentage as the rest of the population.

In the main, however, hereditarily transmissible variations depend, not upon mutation, but upon combination, that is to say upon the variable appearance and disappearance of continually changing factor combinations. For the most part, indeed, we have to do here with combinations which occur in accordance with mendelian rules.

Now we have to ask ourselves what will happen to a hybrid population in the course of time, what will be its composition after a long series of generations? Let us illustrate the matter by a simple instance. Suppose that we have a number of F_1 hybrids between a black (*AA*) and a white (*aa*) race of rabbits. Let us confine a pair of these hybrids (*Aa*) in a large hutch where they can propagate under the most favourable conditions possible. They are to be allowed to breed freely for a number of generations until we have a great stock of rabbits. Now, what will be the composition of such a stock? It will be found to consist of white rabbits and black rabbits in the almost exact ratio of 1 : 3. We had confined a female and a male rabbit of the F_1 generation in the great hutch. Their immediate offspring, those of the F_2 generation, must $\frac{3}{4}$ of them be black rabbits and $\frac{1}{4}$ of them white. Let us suppose that this F_2 generation consists of 8 rabbits in all, these being:

1 homozygotic black male	*AA*,	subsequently to be called male I
2 heterozygotic black males	*Aa*,	subsequently to be called males II and III
1 homozygotic white male	*aa*,	subsequently to be called male IV
1 homozygotic black female	*AA*,	subsequently to be called female α
2 heterozygotic black females	*Aa*,	subsequently to be called females β and γ
1 homozygotic white female	*aa*,	subsequently to be called female δ

Among these rabbits, 16 different couplings are possible, and, being all equally probable, may be assumed to occur with equal frequency, and to produce the following results, as shown in the subjoined table:

Female	$\alpha \times$	male I	will produce	$\frac{4}{4} n\ AA$ animals
,,	$\alpha \times$	,, II	,, ,,	$\frac{2}{4} n\ AA$, $\frac{2}{4} n\ Aa$ animals
,,	$\alpha \times$	,, III	,, ,,	$\frac{2}{4} n\ AA$, $\frac{2}{4} n\ Aa$ animals
,,	$\alpha \times$	,, IV	,, ,,	$\frac{4}{4} n\ AA$ animals
,,	$\beta \times$	,, I	,, ,,	$\frac{2}{4} n\ AA$, $\frac{2}{4} n\ Aa$ animals
,,	$\beta \times$	,, II	,, ,,	$\frac{1}{4} n\ AA$, $\frac{2}{4} n\ Aa$, $\frac{1}{4} n\ aa$ animals
,,	$\beta \times$	,, III	,, ,,	$\frac{1}{4} n\ AA$, $\frac{2}{4} n\ Aa$, $\frac{1}{4} n\ aa$ animals
,,	$\beta \times$	,, IV	,, ,,	$\frac{2}{4} n\ Aa$, $\frac{2}{4} n\ aa$ animals
,,	$\gamma \times$	,, I	,, ,,	$\frac{2}{4} n\ AA$, $\frac{2}{4} n\ Aa$ animals
,,	$\gamma \times$	,, II	,, ,,	$\frac{1}{4} n\ AA$, $\frac{2}{4} n\ Aa$, $\frac{1}{4} n\ aa$ animals
,,	$\gamma \times$	,, III	,, ,,	$\frac{1}{4} n\ AA$, $\frac{2}{4} n\ Aa$, $\frac{1}{4} n\ aa$ animals
,,	$\gamma \times$	,, IV	,, ,,	$\frac{2}{4} n\ Aa$, $\frac{2}{4} n\ aa$ animals
,,	$\delta \times$	,, I	,, ,,	$\frac{4}{4} n\ Aa$ animals
,,	$\delta \times$	,, II	,, ,,	$\frac{2}{4} n\ Aa$, $\frac{2}{4} n\ aa$ animals
,,	$\delta \times$	,, III	,, ,,	$\frac{2}{4} n\ Aa$, $\frac{2}{4} n\ aa$ animals
,,	$\delta \times$	,, IV	,, ,,	$\frac{4}{4} n\ aa$ animals

Total $\frac{16}{4} n\ AA$, $\frac{32}{4} n\ Aa$, $\frac{16}{4}\ aa$ animals
i.e. the ratio will be $4 n\ AA : 8 n\ Aa : 4 n\ aa$ animals

The foregoing table likewise shows what will be the outcome of all 16 possible couplings taken together, and this is that the offspring produced by the three couplings of an F_2 generation consisting of *AA* rabbits, *Aa* rabbits, and *aa* rabbits in the ratio of 1 : 2 : 1, will itself consist of *AA*, *Aa*, and *aa* rabbits in exactly the same proportion—that is to say the F_3 generation thus produced will be of identical composition with the F_2 generation. The same applies to succeeding generations: and it does so even if the animals that couple do not belong to only one generation, but inter-breed promiscuously no matter to which generation they belong—as will actually happen in such an experiment. Similar calculations can be made when the F_1 hybrids are distinguished one from another in a more complicated way by several characters, so that they separate out in the ratio 9:3:3:1, or in the ratio 27:9:9:9:3:3:3:1. Then, too, successive generations will have approximately the same composition as the F_2 generation.[1] When, therefore, any stock continues to increase by free coupling from among the members of an F_2 hybrid generation, this stock will continue to show approximately the same composition as the original F_2 generation, provided always that no selection takes place.

In nature, it will seldom if ever happen that entire populations, such as all the rabbits of one island, are derived from a single F_2 generation of uniform composition, but the law we are considering has far wider applications.

We can, for instance, modify the above-described experiment. Let us, in our great rabbit hutch, confine as progenitors for our new population of rabbits the following animals: 2 homozygotic black *AA* males (named I and II); 2 homozygotic black *AA* females (named α and β); 1 heterozygotic black *Aa* male (named III); and 1 heterozygotic black *Aa* female (named γ). If we allow these six rabbits to couple irregularly as often as they please, we shall get as offspring a generation of rabbits consisting of homozygotic blacks, heterozygotic blacks, and heterozygotic whites, in the ratio 25 : 10 : 1.

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[1] Such a population does not, indeed, remain perfectly constant even when selection is entirely excluded, but it would lead us too far to trace the necessary calculations here.

Calculation gives us the following table:

female	$\alpha \times$	male	I	will produce	$\frac{4}{4}\,n$ black AA animals
,,	$\alpha \times$	,,	II	,, ,,	$\frac{4}{4}\,n$ black AA animals
,,	$\alpha \times$	,,	III	,, ,,	$\frac{2}{4}\,n$ black AA, $\frac{2}{4}\,n$ black Aa animals
,,	$\beta \times$	,,	I	,, ,,	$\frac{4}{4}\,n$ black AA animals
,,	$\beta \times$	,,	II	,, ,,	$\frac{4}{4}\,n$ black AA animals
,,	$\beta \times$	,,	III	,, ,,	$\frac{2}{4}\,n$ black AA, $\frac{2}{4}\,n$ black Aa animals
,,	$\gamma \times$	,,	I	,, ,,	$\frac{2}{4}\,n$ black AA, $\frac{2}{4}\,n$ black Aa animals
,,	$\gamma \times$	,,	II	,, ,,	$\frac{2}{4}\,n$ black AA, $\frac{2}{4}\,n$ black Aa animals
,,	$\gamma \times$	,,	III	,, ,,	$\frac{1}{4}\,n$ black AA, $\frac{2}{4}\,n$ black Aa, $\frac{1}{4}\,n$ white aa animals

The outcome of all the possible pairings will be:

$\frac{25}{4}\,n$ black AA, $\frac{10}{4}\,n$ black Aa, $\frac{1}{4}\,n$ white aa animals

Since all the couplings are equally probable, the outcome of a very great number of such couplings will be a population in which the categories *AA*, *Aa*, and *aa*, are in the ratio of 25 : 10 : 1, that is to say a population having the same composition as the F_1 generation.

If we allow such a population of rabbits to continue breeding promiscuously unchecked, then all the successive generations will exhibit approximately the same numerical ratio between the white and the two kinds of black. It is easy to calculate for any generation we please what the composition will be.

Provided always, of course, that there has been no "admixture of foreign blood", and that the various categories have equally vigorous powers of life and reproduction so that no selection interferes with our results.

But what will happen if some particular combination is persistently kept apart from the promiscuous interbreeding, or is only able to breed less effectively than the other combinations? Once more let us consider a simple instance. Let us stock an island with two female blue Angora rabbits having the formula *AA XX BB CC dd gg vv* (regarding this formula, see above, pp. 58–9) and two male short-haired wild-coloured rabbits having the formula *AA XX BB CC DD GG VV*. The F_1 generation will then consist of —[1] *Dd Gg Vv* rabbits, which are all short-haired and wild-coloured; and the F_2 generation will consist of the following categories in the ratio shown in the last column:

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[1] The — prefixed to the formulas that follow is used for shortness, to denote the homozygotic factors *AA XX BB CC*, which are indifferent to our present consideration.

— *DGV*	wild-coloured short-haired . .	27
— *DGv*	,, ,, Angora . . .	9
— *DgV*	black short-haired . . .	9
— *Dgv*	,, Angora	3
— *dGV*	blue wild-coloured short-haired.	9
— *dGv*	,, ,, ,, Angora .	3
— *dgV*	,, short-haired	3
— *dgv*	,, Angora	1

In the absence of selection, the resulting population of rabbits will, as already said, continue indefinitely to exhibit this multiform composition. But let us now assume that particular categories—say all blue wild-coloured Angora rabbits, that is to say all rabbits having the formula — *dd GG vv* and — *dd Gg vv*—are removed from the promiscuous stock before they have time to breed. Even if this destruction or removal of the blue wild-coloured Angoras were very strictly carried out, if no beast having these characters were ever allowed to take part in the promiscuous breeding, nevertheless such animals would continue to be born in our rabbit population, seeing that beasts having that particular heredity formula and those particular characters would be procreated in such a stock by parents which were outwardly quite different! But already in the first generation after selection has been begun, the number of blue wild-coloured Angoras will be smaller. The following table shows the effect of such an elimination of blue wild-coloured Angoras:

In the absence of selection, the F_2 generation must contain the following categories	in the following ratios	expressed in percentages	the elimination of all blue wild-coloured Angora rabbits gives the following percentages	increase or decrease in the percentage
wild-coloured short-haired . .	27	42·18	44·44	+ 2·26
,, ,, Angora . .	9	14·06	13·82	— 0·24
black short-haired . . .	9	14·06	14·67	+ 0·61
,, Angora	3	4·69	4·46	— 0·23
blue wild-coloured short-haired	9	14·06	13·82	— 0·24
,, ,, ,, Angora .	3	4·69	3·19	— 1·50
blue short-haired	3	4·69	4·46	— 0·23
,, Angora	1	1·56	1·12	— 0·44

A glance at the foregoing table shows (though detailed calculations are not given, since they would bulk too largely) that the composition of the F_1 generation after such a selection would be one in which, not only the frequency of the wild-coloured Angoras would have diminished, but also there would be notable differences in the relative proportions of the other categories.

If such a selection be continued for a long series of generations, rabbits of the eliminated kind will become continually rarer, and in other respects the composition of the rabbit population will be very greatly modified.

In particular experiments, this effect will differ greatly, according as dominant or recessive types are eliminated, according as only one combination is eliminated at a time or several combinations at once, and so on. It would lead us too far afield to consider all the details here. What concerns us is to know that such a process of selection does not lead to the complete disappearance of particular combinations, but that the eliminated types become rarer and rarer, and that in other respects the composition of the population is modified.

Selective processes are of the utmost importance to the preservation and to the further development of a "species". As we have already learned, in all living organisms numerous mutations occur, and the great majority of these are developmental defects. Now, in plants and animals living in a state of nature, all the individuals below a certain level of efficiency are eliminated in the struggle for existence and do not succeed in reproducing their kind. Thus under natural conditions we find a very uniform stock of normal and healthy animals. On the other hand, in the case of every kind of plant or animal that we "domesticate", there soon originates a considerable number of more or less peculiar races. From the wild cabbage (Brassica oleracea) there thus arise the turnip cabbage, the common white cabbage, the cauliflower, Brussels sprouts, etc.; and from the wild rabbit there arise the numerous breeds of tame rabbits. In reality, however, they do not originate as the outcome of domestication, the actual fact being that all they owe to domestication is their preservation. Mutations are continually arising everywhere, but natural selection eliminates all that are not fitted to survive, and thus keeps the type uniform. The most notable effect of domestication is that it puts an end to natural selection. Now, obviously, civilised human beings are in this respect living under similar conditions to domesticated animals. As long ago as the middle of the diluvial period our ancestors, as civilisation slowly increased, were more and more withdrawn from the working of natural selection, and accordingly latter-day mankind shows phenomena of degeneration similar to those which have appeared in all other living creatures withdrawn from the working of natural selection.

Interference with natural selection is injurious, not only because the spontaneously and continually arising low-grade mutations are not eliminated, but also because after every racial crossing a very variously mixed population arises (see above page 100) in which the individual racial differences manifest themselves in all conceivable combinations. Some of these combinations will lead to the production of individuals representing the bad qualities of both races, or qualities that do not harmoniously combine, although, of course, other individuals will embody extremely desirable combinations. Under natural conditions, natural selection would in the end restore such a mishmash to a uniform type, and the one best adapted to the environing conditions. But where selection has ceased to operate effectively, there will be a tendency to the indiscriminate survival of all the combinations, including those of low grade.

Contemporary civilised nations, and also most of the surviving peoples we still class as "primitive", show very clearly the effects of the hindrance to natural selection. Individuals equipped with the satisfactory bodily and mental health which is the rule in wild animals are exceptional in the human species.

Among civilised nations there are superadded to the effects of interference with natural selection the effects of a process of reversed selection which will be discussed later. This operates because, under civilised conditions, the best-equipped human beings reproduce less vigorously than do average members of the population.

In domesticated animals, natural selection is almost always replaced by an artificial selection, by the breeder's choice of particular types to breed from, partly under the guidance of practical considerations, and partly under that of whims (as in the production of pug-dogs, and the like).

In the human species, on the other hand, there is as yet no attempt at such an artificial selection. Owing to the lack of any such process, we do not find in civilised human beings as we find in dogs, pigs, cattle, etc., sharply distinguished racial types brought into existence by the breeder's art, but, instead, a "raceless" mixture resembling that of pariah dogs. Yet it would be very easy, if only men and women could be arbitrarily coupled in accordance with a preconceived plan, to breed human beings into artificial races as clearly distinguishable one from another and as unified in type as fox-hounds, greyhounds, dachshunds, poodles, etc.

To-day it seems almost impossible to doubt that natural selection is a force tending to promote the preservation of species, and that every kind of plant and animal will be prone to degenerate when the working of natural selection is interfered with. There is still, however, plenty of room for divergent opinions upon the question as to how far the natural selection of variations affects the further development, the "evolution", of living beings. What has been said in the foregoing pages will have shown that of the three above-described categories of variations, hereditarily transmissible variations, that is to say only mutations and combinations, can offer material upon which natural selection can work in this direction.

CHAPTER FOUR

EFFECTS OF INBREEDING

When inbreeding is practised in the case of an organism wherein cross-fertilisation is the rule, the results are almost invariably disadvantageous. The recognition of the injuriousness of inbreeding dates from ancient times, so that a considerable part of the legislation of the oldest civilised races turns upon this question.

The bad effects of inbreeding depend upon two very different things. In the first place, inbreeding and reproduction from individuals who are closely akin favours the mendelising-out of recessive developmental defects. To understand how this happens, study the genealogical tree shown in Fig. 25. Albinism is transmitted by inheritance in human beings as a simply recessive character. Heterozygotic albinotic human beings are perfectly normal to outward aspect. If the members of such a family hereditarily tainted with albinism mate with members of a family not so tainted, then some of the offspring of such unions, though not born albinos, will carry the taint

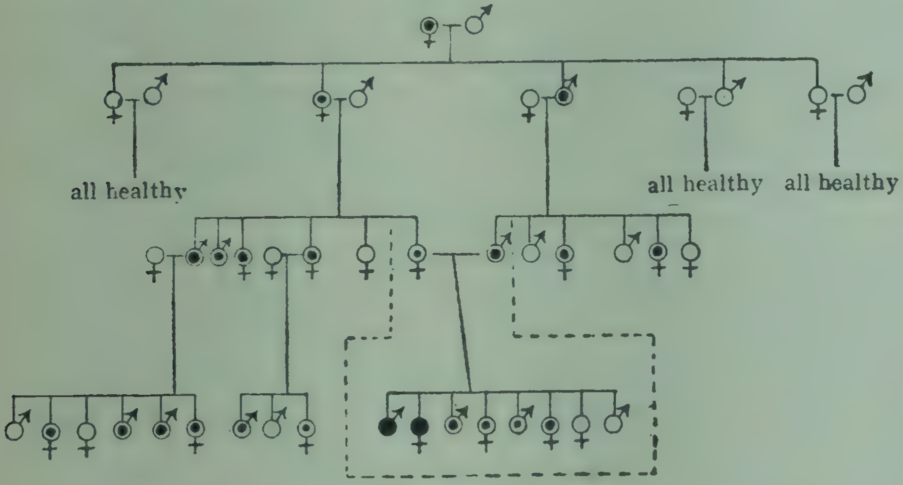

Fig. 25.

Genealogical tree of a family with a recessive mendelising hereditary developmental defect (such as albinism). Homozygotic healthy persons are indicated by white circles; heterozygotic (still outwardly healthy) persons are indicated by white circles with a black point in the centre; homozygotic diseased persons are indicated by black circles. The one marriage of near kin (the parents and their children are marked off from the rest of the table by a dotted line) renders possible the production of homozygotic diseased offspring.

heterozygotically and thus in a latent form. Homozygotic albinos only appear as the offspring of such heterozygotic individuals when two of them have joined in marriage. Obviously, however, the marriage of near kin within such a tainted family will greatly increase the probability of a union being one of this kind, so that the appearance of overt albinism in the family will be greatly favoured by close inbreeding. Many families carry such hereditary taints without the members being aware of it when the taints are recessive. In one family it will be one taint, and in another it will be another. Marriage outside the family will not lead to the appearance of the taint, for the marriage will not produce homozygotic offspring; but marriage within the family will favour its appearance. This is one cause, though not the only one, of the ill-effects of inbreeding.

A second kind of ill-effect from inbreeding depends upon the fact that, for unknown reasons, inbreeding (the more speedily, the closer it is) weakens the offspring and reduces the capacity for reproduction. This weakening always progresses until, sooner or later, a stable minimum is attained; that is to say when inbreeding is practised for many generations the vital efficiency of the offspring decreases at first rapidly generation after generation, but eventually the decrease slackens, and in the end a condition is reached wherein further inbreeding does no more harm. The minimal efficiency thus reached varies greatly from one organism to another.

As to this particular type of ill-effects from long-continued and close inbreeding, we have no trustworthy information in the case of the human species. Very little is known about it, moreover, as regards the higher animals.

Though it is occasionally observed that the offspring of incestuous marriages (the children of a brother and a sister, of a father and a daughter, and so on) are apt to be mentally and physically defective, there seems little reason to suppose that this is due to inbreeding. Speaking generally we may say that to-day incest is likely to occur only in persons who are greatly below par, and history has recorded numerous cases of incest in high-grade families where the children manifested no such signs of degeneration.

SECTION TWO

RACIAL DIFFERENCES IN MANKIND

by

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PRELIMINARY CONSIDERATIONS

When we study the human species as distributed in space and time on the surface of the globe, not only do we find differences everywhere and at all times, but even at the same time and in the same place we never come across two individuals who are exactly alike. Nevertheless, in particular groups the members resemble one another more closely than do the members of two distinct groups. The question now arises as to the significance of these differences between individuals and groups. Does environment play a part? Do, for instance, climate, nutritive conditions, or perhaps the social "milieu", account for these differences? Can the "characters" upon which the likenesses and differences between individuals and groups depend, be modified by changes in such external influences? Or, on the other hand, are there characters which are quite independent of such "peristatic" influences? Are there characters which are part of the hereditary equipment, and therefore inalienable? The science which is concerned with the study of these problems may be termed "anthropobiology". The subject matter of anthropobiology is, then, a study of the individual differences and the group differences (racial differences) between human beings, and an attempt to discover to what extent they are, respectively, "paravariations", "mixovariations", or "idiovariations" (see above, pp. 25 et seq.).

Hitherto systematic anthropology has regarded such racial distinctions almost exclusively from the anatomical standpoint, describing them and measuring them. Of course this anatomical study is indispensable to the science of racial hygiene. But it is no less indispensable that we should know which among these outwardly recognisable characters are hereditarily transmissible. Observation of shapes and sizes does not suffice to tell us this. The characters in question must also be regarded "biologically", must be studied in relation to their origin, and in relation to their significance to individuals and to groups. Biological knowledge as well as anatomical must provide the foundation for racial hygiene.

CHAPTER FIVE

VARIABLE CHARACTERS IN HUMAN BEINGS

(SPECIAL ANTHROPOLOGY AND ANTHROPOBIOLOGY)

IN human beings, all the organs and all the parts of the organs are subject to variations. Negroes and Europeans, for example, are distinguishable from one another, not only by the tint of the skin, the growth of the hair, the shape of the nose, the lips, and the skull, and by other widely recognized differential characters; but also by peculiar differences in the brain, the muscles, and the viscera. If, therefore, we were going to write a complete textbook of anthropology, all the organs and anatomical systems of human beings would have to be fully described.[1] Here, however, I only design to provide the reader with such elementary knowledge as is requisite for the understanding of the racial-hygienic inferences which can be drawn from a knowledge of the laws of inheritance.

1. SKULL

No part of the human body has been more exhaustively studied by anthropologists than the skull. In respect of size and shape, and in all its details, it has been subjected to precise measurements, and elaborate graphic methods have been applied. Here no more than a summary of the most important achievements can be attempted.

For the purposes of anthropological study, the characters of the skull can be divided into groups.

First of all, there are a great many characters, both qualitative and quantitative, whereby monkeys and human beings are plainly and sharply distinguished from one another. These characters, therefore, are specific to human beings and must belong to the human heritage. Anthropoid apes (chimpanzees, gorillas, orangs, and gibbons), for example, are distinguished from men by the small size of the cranium as compared with the facial part of the skull, by the great size of the supra-orbital prominences, by the way in which the canine teeth stand out, and by the marked development of the lower jaw although the chin is receding. The characters of the human skull, which are the precise opposite in all these respects, are unquestionably hereditary—are part of the general human heritage. Occasionally, however, we note apelike or "pithecoid" characters in the human skull. We must assume, therefore, that the common ancestors of human beings and apes possessed like characters, and that now and again, for reasons concerning which we as yet know very little but which we sum up under the name of "atavism", such an ancestral character reappears, just as a dark stripe sometimes appears running down the back of a horse.

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[1] This book is not written for anthropological specialists. Those who need information concerning the details alluded to in the text, and those who are in search of a full description of anthropological technique, may be referred to the admirable, nay indispensable, Lehrbuch der Anthropologie by Rudolf Martin.

Furthermore, when we compare the skulls of contemporary human beings with the skulls of Homo primigenius (Neandertal, Spy, La Chapelle, etc.), with that of Homo heidelbergensis, with that of Pithecanthropus erectus (Java), and these with one another—we note a number of marked differences. Atavism may be displayed in the case of these characters likewise. For instance, now and again particular Neandertal characters may turn up in contemporary human beings, as reversions to the Neandertal or similar ancestral forms; but we never get a reproduction of all the genuine Neandertal characters in combination.

For the purposes of racial hygiene, however, the before-mentioned characters are of less importance than the following.

A great many of the peculiarities of the skull may be regarded as racial characters in the narrower sense. Above all I speak of quantitative differences in the whole skull or in its parts; but also of qualitative differences, which occur in numerous combinations, and thus help us to distinguish between races. Any particular character may naturally occur in various combinations with other characters in very different races. But in the case of each character a special investigation must be made to ascertain whether it is really inheritable, whether it is a "racial character"—for many skull characters common in particular human groups occur only because all members of the group have been exposed to identical determining influences. Such characters are not racial characters.

Among racial differences, the most striking is that which concerns the general shape of the skull, both of the cranium and of the facial part of the skull (including the nose).

In addition to giving verbal descriptions, anthropologists take measurements in order to determine and to record differences in the shape of the skull. Since the practice was first introduced by Anders Retzius (1864), to overcome the difficulty in comparing the length and the breadth of skulls of different sizes, it has been usual to represent two such dimensions in percentage ratios one to another. Every such ratio is spoken of as an "index". Thus the breadth of the skull is recorded in a percentage of the length, and we speak then of the "cranial index"—the index of the breadth and length of the skull. This particular index concerns only the dimension of the brain-box, having nothing to do with the face. (See Fig. 26.) It is necessary to insist, however, that the resulting classification into dolicocephals or long heads, mesocephals or intermediate heads, and brachycephals or broad heads, is arbitrary—at any rate, we are not entitled to assume without further examination that dolicocephals here, and brachycephals there, as determined by measurement, can be classed together in a group. For instance, the relative length of the skull among the long heads in Sweden is determined by quite different peculiarities of bone formation from the long-headedness among certain African Negroes. Dolicocephaly and brachycephaly depend upon details of form in the frontal

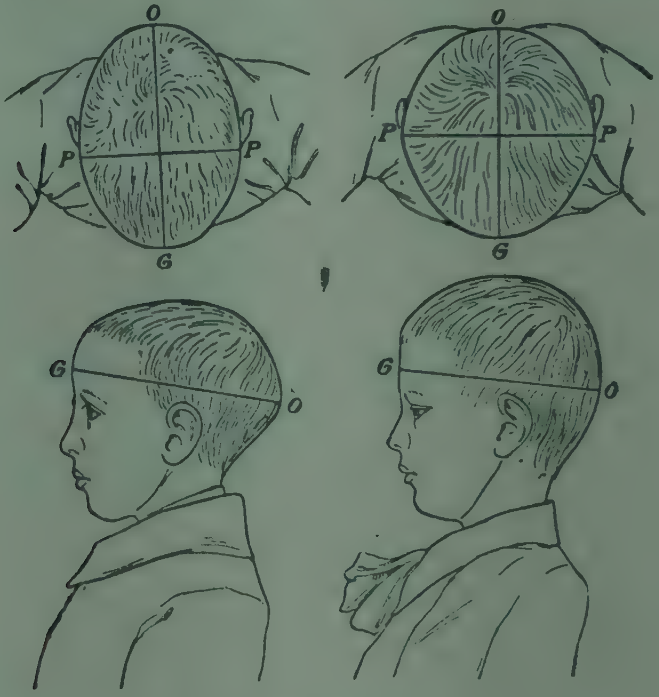

FIG. 26.

Dolicocephalic and brachycephalic skulls.
First boy: length of head 190 mm., width of head 137 mm., cranial index 72·1.
Second boy: length of head 174 mm., width of head 154 mm., cranial index 88·5.
G—O = greatest length of head, P—P = greatest width of head (after Röse).

bones, the parietal bones, the occipital bones, etc. The designations "dolicocephal", etc., merely provide us with a brief expression for the classification of the ratios between two main dimensions of the skull. Above all, it is erroneous to regard the arbitrarily determined limits between long heads, intermediate heads, and short heads or broad heads, as naturally-given racial boundaries. The natural oscillation of every character on either side of a mean, pays no heed to such categories. The shortest skulls belonging to a typically long-skulled race will usually come well within the category of the "mesocephals", but this does not signify that they are inferior in respect of racial purity to the longer skulls.

It is, therefore, utterly mistaken, when from half a dozen to a dozen prehistoric skulls are found and when some of them are mesocephalic and others brachycephalic, to begin forthwith to speak of racial admixture. The great probability is that they are only varying specimens of one and the same race. Seeing that a race may have its average cranial index just where, according to the traditional classification in these matters, one category shades into the other, pedantic systematists might declare that such a race exhibits two different skull-shapes, when in reality it is just as homogeneous as any other!

Speaking generally, the skulls of every race manifest a large number of plainly recognisable characters peculiar to that race. For instance, it is easy enough to distinguish a narrow, long, and squat Negro skull, with its marked frontal and parietal eminences, with its flat and small nasal bones, its conspicuous pre-nasal fossae, its low, round, nasal orifices, its low-arched front, its markedly prognathous character, its thick zygomata, etc.—from the large, strongly arched, long, narrow, "Nordic" skull, with its prominent, long, and narrow nasal bones, set each against the other like the sides of a roof, with its upright "orthognathous" profile, its long and narrow face, its strongly developed chin, etc. This signifies that in all these respects we have to do with characters which form part of the hereditary equipment of the races concerned, and are therefore part of a fixed inheritance. Nevertheless, the outward aspect of any of these characters may have been altered by "peristatic" influences, that is to say by the effect of the environment.

The racially characteristic, the hereditary form of the skull, such as the cranial index, is already manifest in the foetus during the last months of intra-uterine life, although such racial distinctions are by no means so conspicuous then as they become later.

The pressure undergone by the skull during birth has the result, as is well known, of modifying its shape. During birth, the edges of the cranial bones overlap, and they are bent in various ways. When the presentation is abnormal, such moulding of the head is especially marked. In most cases, these effects are not permanent, but pass away without leaving a trace after a few days, weeks, or months. In exceptional instances, permanent malformations of the skull may arise at these times, but such pathological forms need not here be considered.

If, as is normally the case, the child's head, as it continues to grow after birth, acquires the form proper to its ancestry, becoming more typically racial, this is generally assumed to be the effect of a hereditary trend of growth. It is supposed that the individual parts of the skull must grow with a definite intensity and speed, and must develop in a particular order. This growth is, however, directly regulated by the internal secretions of the glands, by the "incretions", the "hormones". The most important of the endocrine glands in this connexion is the pituitary; and next in importance come the reproductive glands. In boys who have been castrated, the cartilaginous areas between the sphenoid and the occipital bones are unusually slow to ossify. Thus every change in endocrine functioning modifies the growth of the skull and thereby affects its shape. We do not know how far, in normal life, the endocrine functions can be modified by external influences. If such modifications be possible, then the shape of the skull can be changed by external influences.

Under morbid conditions, such results ensue. In the disease known as acromegaly, the outcome of disease of the pituitary, there occurs, in addition to an abnormal growth in length of the fingers and the toes to as much as twice the normal, an excessive growth of the lower jawbone. In consequence of this, the chin is unduly developed, and projects in front of the upper jaw. This malformation is known as "progenia". A shortening of the facial part of the skull is normal in domesticated swine, and is generally upposed to be the outcome of changes in the pituitary incretion.

(Fröhlich.) Here, then, peristatic influences are plainly at work; and in the case of many of the characters of the skull it is difficult to say how far the peculiarities are due to peristatic influences, and how far they are hereditary. In the instance above given, it is the lower jaw which is modified, but, under other conditions, similar influences may affect the whole shape of the skull. It is a familiar fact that the morbid growth of the bones characteristic of rickets may lead to markedly pathological forms of skull (caput quadratum). But even within the domain of the normal, the head may very well be affected in its shape by the varying speed of the growth of the brain, by individual differences in the thickness or the superficial extent of the individual bones, and by other such influences.

The growing head can also be moulded in various ways by external influences. I need mention only in passing such artificial deformities of the skull as are produced by some of the Indian tribes and by certain other races, the heads of infants being confined between boards or tightly bandaged. Very tight cap-ribbons (Heligoland) may have a similar effect. Slight differences in the form of the skull may even be brought about by keeping the infant habitually lying upon something hard or upon something soft, as the case may be. The heads of mon-oval twins,[1] which in other respects are extraordinarily alike (see below, pp. 124 et seq.), may in this way be modified or "deformed" to an extent amounting nearly to 8 points of the cranial index.[2]

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[1] "Mon-oval" twins, are twins originating out of a single ovum which has abnormally divided into two halves, so that an entire individual grows out of each half. Such twins obviously have an absolutely identical hereditary equipment. One and the same hereditary equipment is here assigned to two distinct persons, who are hereditarily the most closely similar of all the living organisms that are produced by sexual reproduction. Unfortunately, however, it is not invariably easy to determine whether we have to do with mon-oval twins; for even when the condition of the membranes has been carefully noted at birth, this point is not always decisive. The diagnosis is placed on a sure foundation when the twins resemble one another very closely in every detail. Experience has shown that the following points are the most important in this respect: the colour of the hair, including its tendency to turn white; the general distribution of the hair on the head and on the body, the lanugo; the colour of the eyes, including the structure of the iris; the tint of the skin; the supply of blood to the skin; freckles; folds on the tongue; the shape of the face, the ears, the hands, the finger-nails; the general build of the body.

[2] Walcher, Weitere Erfahrungen in der willkürlichen Beeinflussung der Form des kindlichen Schädels, "Münchener Medizinische Wochenschrift", 1911.

Far more interesting than these crude effects, whose method of production is easy to understand, are the observations that have been made upon changes occurring in the shape of the skull among races which have left their homes and have migrated into a new environment.

In the children of Jews who have emigrated from eastern or central Europe to the United States, the skulls are narrower than those of their broad-skulled parents, and this comparative narrowness is more marked in proportion to the number of years that have elapsed since the migration. Conversely, the children of dolicocephalic Sicilians acquire somewhat broader heads in the United States. The same phenomenon has also been noted among the children of dolicocephalic Scots, who become more brachycephalic. Again, children of Spanish blood born in Porto Rico have rounder heads than their parents.[1] Observations made in Germany likewise show that environmental influences have a direct effect upon the shape of the head. In certain parts of the Black Forest, and in the Grosse Walsertal in Vorarlberg, more than 98 % of the population consists of round-heads, of whom one-fourth have light-coloured eyes and fair hair. It is impossible to account for these conditions on the hypothesis of selection, produced perhaps by emigration, seeing that with the elimination of the dolicocephals there must have been a diminution of the fair-haired and pale-eyed members of the population; for, in view of the primitive racial inter-connexion between the respective characters, it seems impossible that nearly all the dolicocephals could have been removed without the removal of any of the blonds.[2] These observations in the United States and in German-speaking lands can only be explained on the hypothesis that in particular regions certain environmental influences can directly or indirectly modify the shape of the skull. As to the nature of such influences we know nothing.

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[1] F. Boas. Changes in Bodily Form of Descendants of Immigrants, Washington, 1910 and 1911—The Anthropometry of Porto Rico, "American Journal of Anthropology", III, 1920.—Guthe, Notes on the Cephalic Index of Russian Jews in Boston, Ibid., I, 1918.—Fischer, Betrachtungen über die Schädelform, "Zeitschrift Morph. Anthr.", vol. XXIV. 1924.

[2] Even if we assumed that the aborigines of these regions were two brachycephalic races (the Alpine and the Dinaric, see below), both dark, this does not explain the non-disappearance of the blonds and the disappearance of the dolicocephals.

I may allude, however, to a pioneer experiment attempting to elucidate these influences. Experiments have been made on rats, in which vitamin deficiency both before and after birth appeared to cause changes alike in the shape and in the size of the skull. These purely chemical influences modified the skulls plainly in the direction of brachycephaly.[1] Injections of thallium caused similar changes.

On no account, however, must we forget that all such observations and experiments show nothing more than this, that certain elements in the shape of the skull (as in the stature in the instance given below, pp. 150 et seq.) are modified by environmental influences—all the rest remains part of the inalienable hereditary equipment. A delimitation of the two spheres is often impossible.

Finally there must be mentioned the last of the known influences of external factors upon the shape of the skull, the influence being in this case an indirect one exercised through the stature. With increasing stature, the growth in length of the skull becomes somewhat more rapid in proportion to the growth in width, with the result that the cranial index (the ratio of the width to the length) becomes somewhat smaller. Now, as shown below (pp. 150 et seq.), if certain pathological cases be left out of consideration the growth of the individual in height is to some extent influenced by nutritive conditions, and perhaps by other circumstances of the environment, during the period of growth. Thus according as these influences make the stature greater or less, there will be a corresponding decrease or increase in the cranial index. When considering the significance of this index in any individual, therefore, we must take the stature into account.

The conditions of life can also, in some measure, affect the growth of the facial elements of the skull. When we compare

[1] E. Fischer, Schädelform und Vererbung, "Münchener Medizinische Wochenschrift", 1923, no. 50, and "Zeitschrift Indukt. Abst. und Vererbung," no. 33, 1924.—Neubauer, Experimentelle Untersuchungen über die Beeinflussung der Schädelform, "Zeitschrift Morph. Anthr.", vol. XXIII., 1925.

the socially higher and the socially lower strata of the population in the same race, we find that in the former the face is on the average finer and narrower, and in the latter coarser and wider. We see this among the Germans, and we see it likewise in Japan. The difference depends, not only upon the soft parts, but also upon the structure of the bones. Such differences, however, are very slight, and we must be careful not to confuse them with the genuinely racial distinctions which often exist in one and the same population, the racial composition of the socially higher and socially lower strata of the population being distinct. The latter fact has long been known; but, of late, scientific work has been done to elucidate it, and the differences in question between the two strata have been described as "constitutional differences". In Japan, for instance, Baelz has made such an investigation.

The function of speech, likewise, can influence the shape of the lower half of the face, through the pull of the masticatory and facial muscles upon the growing jaw bones. In the Americans, and to some extent in the English, there are fairly plain indications of this; and in South Germany it is perhaps possible to detect slight differences between the Franconians and the Alemanni.[1]

Finally I may mention that the sexual differences in the skull, which are common to all races, are determined by the internal secretions of the reproductive glands.

As regards the craniological characters considered above, we can always trace the working of non-hereditary "peristatic" factors, which have been influential to a varying degree. But these numerous instances of the widespread effects of environmental influences, unmistakable though they are, must not lead us to underestimate or even to overlook the outstanding power of hereditary forces in moulding the skull. Careful observation convinces us that the hereditary equipment is of supreme importance in this respect. All the modifying influences act upon the foundation of a capacity to develop in a definite direction, to a definite size or shape. The effects of environmental conditions are only, as it were, lightly sketched in upon what may be called an ideal plan of development. We find whole groups of human beings among whom thousands upon thousands of individuals living under very various conditions can be shown, on close investigation, to have the same shape of skull, which persists as a definite racial form. This, of course, does not mean that the shape is absolutely identical in all such persons, but that it varies regularly on either side of a typical mean. Thus each race has a typical cranial index. The peculiar distribution of the cranial index throughout the world, or its distribution in Europe separately considered, serves to show that this index is a racial character, and that it is determined by race. Nothing else can explain the details of its distribution (see below, the subsection on European anthropography). The tenacious way in which the racial form of the head continues to manifest itself amid the before-mentioned modifications is especially plain when we can study its emergence during long periods of history and in prehistoric times, throughout the vicissitudes and migrations of various peoples, and amid the rise and fall of States. In Egypt, for instance, the constancy of a particular form of head can be traced. In the Near East it will be possible to show that in remote areas, isolated by mountains, deserts, swamps, etc., the ancient types have been inherited unchanged for thousands of years, during periods when immigrants into the neighbourhood have been repeatedly eliminated so that the old race has been perpetually reconstituted. That is why it is necessary to stress the teachings of experience. If we have found that certain changes in the length or the breadth or the height of the cranium are peristatically determined, are, that is to say, produced as modifications by the working of environmental forces, this has only served to demonstrate once more the fundamental truth that the basic shape of the skull, its essential form, is inheritable, is fixed and inalterable.

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[1] Hellpach, Das fränkische Gesicht, "Sitzungsberichten Heidelberg. Akad.", section II, 1921.

It seems almost absurd to point out that when a skull has had its index modified from 74 to 76 by the effect of nutrition during childhood or by the way in which the infant was handled or by some other external influence, the person to whom the skull belongs does not become of a different race because he must now be classed as mesocephalic instead of dolicocephalic! Yet it is necessary to say this, for there are still people who regard such terms in a hidebound way, and use the rigidly defined notion of brachycephaly or dolicocephaly to decide the racial appurtenance of a skull.

What has been said above concerning the shape of the cranium applies, mutatis mutandis, to the shape of the face, to the shape of the orbital cavities, the nasal cavities, the hard palate, etc. (For details concerning these matters, as far as the facial bones are concerned, see below.) They, too, are all in the main hereditarily determined. In their case, likewise, the study of races, and especially of isolated races or stocks like the Australian Blackfellows, the Hottentots, etc., supplies us with such typical instances of the racial distribution of particular shapes that it is no longer possible to doubt their hereditary nature.

In these circumstances we cannot be surprised to find that craniometry shows the heads of mon-oval twins to be extraordinarily alike. In two such twin girls, Jewesses, twenty-three years of age, I obtained the following measurements:

	1 Greatest length of head[1]	3 Greatest width of head	4 Narrowest frontal width	6 Zygomatic width	8 Width of lower jaw	18 Height of face	21 Height of nose	13 Width of nose
I	190	150	107	130	89	106	48	30
II	191	149	107	130	91	106	47	30

The cranial indexes of these two girls were respectively 78·5 and 78·9, the absolute differences between the length and the breadth of the skull in the two being only 1 mm. (figures determined by very careful and repeated measurement), while the narrowest frontal width, the greatest facial width, and the height of the face, were identical to fractions of a millimetre! This practical identity could only be achieved by inheritance, for no environmental influences could possibly act so perfectly alike upon two individuals. The greatest difference was that in the width of the lower jaw in the two girls, and we can easily imagine that it is here that external influences must have been most effective. It is true that even in mon-oval twins we sometimes find considerable differences in the cranial measurements,[1] which is not to be wondered at in view of the differences of environment as concerns nutrition (affecting the size), position, etc., beginning already within the mother's womb. Speaking generally, however, we cannot but be surprised to find how little, in these mon-oval twins, external influences have been able to modify the shape of the skull.

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[1] The numbers before the column-heads are those of Martin's method, this observer's technique being used. Measurements in millimetres.

The most convincing proof of the hereditary transmission of these characters would be if they could be shown to mendelise. Now, this seems in fact to happen. Precise demonstration is, however, extremely difficult to secure. From what has been said above, we know that external influences can modify all of the characters to some degree, and we must therefore expect trifling deviations in the cranial index which will interfere with the proper mendelian ratios. Nevertheless, the mendelising of such characters has been shown to be highly probable. First of all, we find that in a typically demonstrable mingling of two different races, the mixed population does not exhibit one single form of skull, with a cranial index oscillating on either side of one mean value, for the curve of variation has two summits, the two original values remaining recognisable. This is what happens in the case of Indian half-breeds in the coastal areas of the United States of America;[2] also among the half-breeds of Damara Land and Great Namaqua Land; and in many other places. The same thing can be established indirectly. For example, Lower Saxony was peopled in the days of the folk migrations before history began. Throughout all the intervening centuries, no uniform intermediate type has come into existence, but the old forms have continued to mendelise out. Nothing but the assumption that particular forms do thus mendelise, and that some of them are unmistakably dominant, will adequately explain the extant forms and the ratios in which they appear.[1]

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[1] Von Verschuer, Die Wirkung der Umwelt auf die anthropologischen Merkmale nach Untersuchungen an eineiigen Zwillingen, "Arch. Rass. Ges. Biol.", vol. XVII.—This work contains an excellent collection of careful observations. The measurements of the trunk and the shape of the head are found to be modifiable to a considerable extent by environmental influences; but the shape of the ear, the nose, and the eye, are very little affected by such influences. (See also Dahlberg in the bibliography.)

[2] Boas, Zur Anthropologie der nordamerik. Indianer, "Zeitschrift Ethnol.", 1895, no. 27.

Nor are direct investigations lacking as concerns the shape of the skull in grandparents, parents, and children. Extremely laborious researches have been undertaken, dealing with extensive material.[2] (The researches related to about four hundred Dutch families consisting of more than three and a half thousand individuals [Frets], to fifty Norwegian families [Schreiner], to almost all [40] the families of the Finnish island Runö [Hildén], and to Norwegian families [Bryn].)

The general results were as follows. There can no longer be any doubt that the development of the shape of the skull is controlled by the mendelian laws of inheritance. There must be a number of cooperating heredity factors at work. (This is in line with Nillson-Ehle's polymeric theory—see above, pp. 61–1.) The reports vary as to whether the factors work in isolation upon the length and upon the breadth of the skull, or whether they influence the general shape as such. Really, we ought not to represent the matter to ourselves so simply as in terms of "size" and mathematical "shape". We have to do with complicated processes of growth during which addition and subtraction of substance occur in numerous bones and in many soft parts. Brachycephaly is dominant over mesocephaly, and mesocephaly is dominant over dolicocephaly. The factors *AA*, *BB*, *CC*, etc., seem to determine the more markedly brachycephalic forms, and the factors *aa*, *bb*, *cc*, the more markedly dolicocephalic forms, while the intermediate forms are heterozygotic. But here, too, the matter is less simple than the foregoing statement implies. There are certainly factors which work in particular directions. We see, for example, in children of the same parents, the quaintest resemblance in the shape and size of the forehead, the skull, and the occipital region, so that there can be no doubt that these characters have been inherited—and nevertheless the cranial indexes diverge considerably (Schreiner). Besides, it seems probable that the extent to which brachycephaly or dolicocephaly manifests itself may depend to some extent upon the absolute size of the head (Frets). Finally, in these family investigations, we once more find plain evidence of the effect of non-inheritable external influences. Not until all the processes of the growth of the skull have been thoroughly investigated, shall we be able to proceed to a more precise analysis of the heredity factors that affect the shape of the skull. Meanwhile we must content ourselves with the knowledge that the mendelian hereditary transmission of the shape of the skull has been proved to occur, so that we know the shape of this organ to be a racial character.

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[1] Hauschild, Die Göttinger Gräberschädel, "Zeitschrift Morph. Anthr.", vol. XXI, 1921, and vol. XXV, 1925.

[2] Frets, Heredity of the Cephalic Index, The Hague, 1924. (See also "Genetica", II–IV, etc.)—Schreiner, Zur Erblichkeit der Kopfform, "Genetica", V, 1923.—Hildén, Zur Kenntniss der menschlichen Kopfform in genetischer Hinsicht, "Hereditas", VI, 1925.—Bryn, The Genetic Relation of Index Cephalicus, "Hereditas", I, 1920.—Vidensk, Skr. math. Kl. no. 5, Christiania, 1921.

The finest outcome of the before-mentioned researches, with their demonstration of a mendelian inheritance of the shape of the skull, is that they explain why, after thousands of years of the mingling of races in Europe and among the offspring of emigrant Europeans in other parts of the world, there has not been produced a population with an average shape of skull, but that instead we find everywhere to-day, as of old, dolicocephals, mesocephals, and brachycephals.

As regards the width of the face, too, there is considerable evidence to show that mendelian rules apply.

Although the Red Indians have broad faces and the white inhabitants of the United States have narrow faces, persons of mixed Indian and white descent in various degrees do not show an average breadth of face, but incline now to one and now to the other parental form. We find the same thing in crosses between the Hottentots and the Boers. There is no question of a mingling of characters or of the production of one single form such as could only be due to environmental influences; both the parental forms continue to exist side by side. But there are complications. It seems plain that the crossing of races has a tendency to lengthen the face somewhat.[1] (See above, pp. 66–7, and below, p. 175.)

The fronto-zygomatic index, the ratio between the width of the forehead and the width of the cheekbones, likewise exhibits mendelian separation in African half-breeds (see second entry under Fischer in the bibliography). Although it has not yet been possible to furnish numerically substantiated proofs that mendelian laws apply to this fronto-zygomatic index as they apply to the colours and shapes of flowers, to the character of the hair, etc., nevertheless, the whole behaviour of this index in peoples of mixed stock, the complete absence of equated intermediate values, the way in which the hybrid values overlap the parental values, and the continued reappearance of the parental values, plainly show the index in question to be a hereditarily determined character. We may then regard it as definitively proved that the main elements in the shape of the cranium and the bones of the face are hereditarily determined and inalterable, and that nothing more than a moderate plus or minus in the measurements is "parakinetic", is "peristatic", is determined by external influences.

2. SKELETON (APART FROM SKULL)

All the separate bones of the skeleton display numerous variations, both racial and individual, as to which I cannot attempt to give details here. Most of the bones certainly have a very marked parakinetic component. The ponderousness of the bones of civilised human beings as compared with those of so-called savages must be mainly the outcome of nutritive conditions. In races whose members are prone to adopt a squatting posture, the femora and the tibiae are apt to be somewhat bent and to be thickened from before backwards; these changes are presumably due to functional causes and are not hereditary. The same thing probably applies to various other characters whose actual cause is still unknown, as for instance to the enlargement of the anterior aspect of the lumbar vertebrae in Europeans as compared with Negroes or Australian Blackfellows. On the other hand, the differences in the shape and the size of the pelvis noticeable as we pass from one race to another are almost certainly hereditary. Wingate Todd has been able to demonstrate the existence of differences in the growth and development of the pelvis in mulattoes as compared with persons of pure white and of pure African descent respectively. ("Am. Jour. Phys. Anthr.", IV, 1921.)

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[1] Lundborg, Die Rassenmischung als Ursache zu auffälligen morph. Veränderungen im Gesichtstypus, Upsala Läkareför. förhandl., 26, 1921.

3. MUSCULAR SYSTEM

In the case of some of the numerous variations which occur in the muscular system, it has been possible to show that they occur with varying frequency in different races, but it still remains uncertain whether they are hereditary. A strong indication of their hereditary character is, however, given by the fact that there are very distinct racial differences in the variations of the palmaris longus muscle in whites, Negroes, Indians, and Japanese, respectively; and by the fact that the absence of this muscle in certain individuals recurs more frequently in their children and in their parents than in individuals belonging to other families.[1] Other differences, some of which extend into the minute structure of the muscles (the cell nuclei), tend to confirm this belief.[2]

4. INTERNAL ORGANS

Like considerations apply to most of the differences which have been found to exist in respect of the frequency of certain structures in the digestive, respiratory, circulatory, urinary, and reproductive apparatus. Differences in the cutting of the wisdom teeth, in the cusps of the teeth, in the number and arrangement of the lingual papillae, in the folds and furrows of the tongue, in the size of the lungs, in the length of the intestines, and many other characters, come under this head. For instance, the absence or the atrophy of the upper lateral incisors has been so often observed to run in families that we have good ground for assuming this to be a hereditary character. As regards the other structures, we still lack definite information.

5. NERVOUS SYSTEM AND SENSE ORGANS

Brains differ very widely in their functioning according to the degree of civilisation of their possessors, and therefore this organ has always been a centre of anthropological interest. As the anatomical expression of the differences in question, we find that there is a marked difference in the average weight of the brain in various human groups. Allowing for differences in stature, which affect the matter somewhat, we find that the average weight of the brain is considerably greater in Europeans, Chinese, and Japanese, than in Negroes and Australian Blackfellows. Within any one race, we find on the average (despite a number of individual exceptions) that persons with high intellectual capacities have heavier brains than the average of their national stock. The convolutions and the furrows or solci between them vary so much from individual to individual that no racial distinctions can be ascertained. (The same remarks apply to monkeys, as for instance to chimpanzees.) We have no certain information as to the hereditary transmission of particular arrangements of the convolutions, though attempts have been made to study this by comparison of the brain of mothers and children, of brothers and sisters, etc.

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[1] Thompson, McBatts, and Danforth, Heredity and Racial Variation in the Musculus palmaris longus, "Am. Jour. Phys. Anthr.", IV, 1921.

[2] Schiefferdecker, "Biol. Zentralbl.", III, 1919.

There can be no doubt whatever that mental aptitudes and capacities are inheritable, the circumstances of life then tending to help or to hinder the development of these aptitudes (see below, Section V). In the present state of our knowledge, we can hardly expect to discern in the brain hereditary differences corresponding to this inheritance of faculty.

As concerns the sense organs, the following facts have been noted. The organ of smell is in human beings in a condition of marked retrogression, as is already the case in monkeys; but, on the other hand, the outwardly conspicuous nose is a typically human structure. The nose differs considerably from race to race, and must be regarded as one of the best racial characters, inasmuch as its numerous variations are, so far as we can judge, purely hereditary. (This matter is more fully discussed below, sub-section 13, Physiognomy.) In the nervous apparatus of the eye we also find racial differences. The inhabitants of Eastern Asia have a flatter fovea centralis and a wider extension of the rod-free zone. The cones are finest and most thickly set in Hottentots, Indians coming next, and Malays next after that. In Melanesians, the cones are less fine, and still less fine in Europeans. These differences must certainly be hereditary.

Among the variations in the eye, especial attention has been paid to the varying colour of the iris (see below, pp. 142 et seq.).

In the apparatus of the eyelids, we are especially interested in "almond-shaped" eyes, those in which the palpebral fissure has a rounded medial and a pointed lateral canthus—in contradistinction to the commoner and more general spindle-shape in which the medial and the lateral canthus are alike. This shape of eye is inherited, being most common in the Near East, recurring from generation to generation, as for instance in the Jewish population. Usually this almond-shape of the eye is associated with an obliquity of the palpebral fissure, so that the outer canthus is somewhat higher than the inner. We often find this, for instance, in Hottentots. The character has been found to mendelise, as it does in the half-castes between Hottentots and Europeans. "Oblique" eyes are recessive to "straight" eyes.

As a malformation we encounter a little web of skin at the inner angle of the eye, forming a fold connecting the upper lid and the lower; this is known as the epicanthus. It has no great anthropological interest, but it resembles (and has been confused with) a typical racial formation, the so-called "Mongolian fold", a variety characteristic of the Chinese, Japanese, Kalmuks (the Mongoloids, in short), and of Eskimos and Malays.

The "Mongolian fold" is a loose fold of skin stretching in front of the upper lid from the side of the nose towards the middle of the eye and gradually disappearing as it passes outwards. It thus hides the inner canthus and the lachrymal papillae, but it never extends so far outwards as to hide the outer canthus. However, it gives the eye a new inner angle, between the edge of the fold and the border of the lower lid, at a rather lower level than the normal canthus, with the result that the palpebral fissure appears to be directed obliquely upwards (the "oblique eyes" of the Mongols), and at the same time the palpebral fissure is very much narrowed towards the nose ("slit-shaped eyes"). If with the fingers we draw the skin of the upper lid upwards, the eye and the lid assume an appearance exactly like those of a European. Since an admixture of Mongolian blood extends as far as Central Europe, it is not surprising that from time to time we note the existence of this fold among members of our own nation, and that it is manifestly hereditary. There is an identical structure in the eyes of the Hottentots, but there can be no question here of any hereditary connexion with the Mongols. In racial crossings, the formation of this fold seems to be a dominant character, but no systematic study of the point has hitherto been made.[1]

In the ear we are interested in cases of an arrest of development at stages which evolution has generally surpassed. Thus we may find that the posterior margin of the external ear is not curved forward as it should normally be; that it exhibits an actual tip, instead of the little Darwinian tubercle; and so on. A crenellated margin to the external ear is peculiar to Bushmen, and is manifestly inheritable. The differences in the shape and size of the ears among our own people are determined by a considerable number of heredity factors. There seems to be a simple pair of heredity factors determining whether the lobule of the ear shall be free or adherent. The character "freeness" is dominant.[2]

6. PIGMENTARY CONDITIONS

The tint of the skin is mainly determined by the presence of little grains of pigment in the epidermis. The less pigment there is, the more does the sanguine tint of the dermis (which is richly supplied with blood) shine through, thus producing the "flesh-colour" of the "white" Europeans.[3] In all races, the pigment grains are situated in the deepest layers of the epidermic cells, but their abundance and their tint vary greatly from one race to another.

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[1] Salaman, Heredity and the Jew, "Journal of Genetics," 1911—I know of a case in which a moderately well-marked Mongolian fold could be traced for three generations in Hamburg. The course of the inheritance suggested here, likewise, that the character was dominant.

[2] Hildén, Ueber die Form des Ohrläppchens, etc., "Hereditas", III, 1922. —Carrière, Ueber erbliche Ohrformen, etc., "Zeitschrift ind. Abst.", 1922.

[3] People of noble birth are often spoken of as "blue-blooded". Actually, in fair and thin-skinned persons of Nordic race, the bloodvessels of the dermis show through with a bluish sheen.

Pigment begins to appear in the skin during intra-uterine life, but at birth the process of pigmentation is far from being completed. A newborn Negro infant is not, like its parents, coloured a brown so intense as to be almost black, but has to begin with very little colour, so that the skin is a slaty-grey or a dirtyish whitey-brown. In newborn infants of the white races, the skin is of a dark-red colour being very richly supplied with blood. The racial equipment of the skin with pigment is completed during the first months after birth.

In addition to the before-mentioned pigment, the human embryo has scattered pigment cells in the corium or cutis vera, these being somewhat more abundant in the skin of the back than elsewhere. In all the Mongoloids, in the Eskimos, the Indians, and many of the South-Sea islanders, the pigment cells are so abundant towards the lower end of the spine, just above the gluteal fissure, that a pigmented patch is formed in the skin, ranging in size from a farthing to a crown piece, or even to that of a small plate. Since the brown pigment is deeply situated, it shows through as of a bluish colour, being known as the "blue spot" or "Mongolian spot". This patch of pigment disappears completely during the first year of life. We must assume that there are specific heredity factors for the production of the Mongolian spot. For this reason we find it from time to time in European children in regions where there is an admixture of Mongolian blood. It has been observed in Hungary, Moravia, and Lower Austria.[1]

The pigment grains of various races seem to be different. Sometimes, indeed, a darker tint of the skin may merely be due to the presence of the same kind of pigment grains in greater numbers. In other cases, however, there is unquestionably a qualitative difference. The Mongoloids have a yellow or yellowish-red pigment. The Red Indians and the Eskimos are like them in this respect. Some races obviously have darker pigment than this. It is possible that the Negro has two pigments. When Negroes cross with whites and the resulting offspring of mixed blood are then crossed with whites again for several generations, we find that the lunulae at the roots of the thumb-nails and in some cases also the skin of the naso-labial fold remain of a yellow tint, although no sign of brownish pigmentation is left anywhere. This indicates the existence of two different pigments for brown and yellow respectively. The racial differentiation of the skin pigment, which obviously presents very various chemical compositions, will doubtless account for the great complication in the tinting of the skin in mixed breeds.

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[1] Adachi, Hautpigment beim Menschen und bei den Affen, "Zeitschr. Morph. Anthr.", VI, 1903.—Zarfl, Ueber die morphologische Bedeutung der blauen Geburtsflecke, "Zeitschrift für Kinderheilkunde", vol. XXXI, 1921.—It will be seen that Zarfl calls the Mongolian spot the "blue birthmark".

We cannot here undertake a comparison of the pigment of the corium and of the tinting of the epidermis in human beings with the pigmentation of the skin in monkeys, but it may be mentioned in passing that monkeys' skin is very variously tinted, even within the same species, this contrasting with the conditions that obtain in other mammals living in the wild state (Toldt Jr., "Mitteilg. Anthr. Ges.", Vienna, 1921).

The question now arises in what way this picture may be modified by non-hereditary factors. The chief of such influences is sunlight, and especially ultra-violet rays. When the skin is exposed to these rays, the epidermic cells are stimulated to produce larger quantities of pigment. The capacity to react in this way varies greatly from race to race in accordance with hereditary peculiarities. In almost all races, the stimulus of sunlight (or of "artificial sunlight") leads to the speedy appearance of a uniformly distributed pigmentation, showing itself in a way characteristic of the race with which we are concerned, but serving in any case to protect the underlying tissue, the vascular network, from the injurious radiations. Such protection is, of course, only relative. The sunburned skin of the southern Europeans does not protect them against the tropical sun as effectively as Negroes are protected by their naturally black skin! In blond whites of the Nordic race, on the other hand, this reaction is almost wholly lacking, and takes place, if at all, very slowly. Consequently, in such persons, the rays of the sun produce their injurious effects on the skin unhindered; the inflammation known as sunburn (erythema solare) ensues; and the injured skin exfoliates in strips. Not until after prolonged and frequent exposure to sunlight does such a skin develop a slight bronzing.

In all races alike, this acquired pigmentation disappears when the exciting cause, sunlight, ceases to act. The sunburned European becomes pallid again in winter; and we find that even the Negro, when removed from the tropics, becomes obviously less dark than he was in his native land.

Other external influences on the pigmentation of the skin are of trifling importance. There may be mentioned here the effect of long-continued heat (blast-furnaces, hot baths, poultices, artificially produced ultra-violet rays, and so on), the transient pigmentation of the skin of the face in pregnant women, the "bronzed skin" of Addison's disease.

What we call the "colour" of the human skin is, then, the product of two factors: first, the hereditary pigmentation; and, secondly, the extant amount of reaction to the effect of light. The paratypical tinting thus modifies the idiotypical. Consequently there is a source of fallacy in comparing the artificially blanched skin of the habitually clad European with that of a "coloured" individual who is accustomed to go naked.

There can be no question about the inheritance of the tint of the skin, even though we are not yet fully informed concerning details. The character is certainly one which mendelises. When crossings take place between light and dark, we do not get a stock of medium tone which breeds true for generations. The cross between a white and a Negro is, indeed, in the first generation a mulatto of medium tint. But the offspring of such mulattoes assort themselves. According to recent and precise researches in Jamaica and Bermuda, we have reasons to suppose that at least two heredity factors combine to determine the brown component in the Negro's skin, and that an additional heredity factor determines the yellow tint. Combinations of these various factors mainly account for the marked variations in the tint of persons whose blood is mixed in varying degrees, and in the offspring of recrossings. Probably, however, additional heredity factors are at work—those that influence the thickness of the skin, etc. Though it is commonly asserted that all mulattoes are of an intermediate brown skin, the before-mentioned studies in Jamaica and Bermuda (relating to one hundred genealogical trees) show the statement to be erroneous.[1] The inheritance of skin-tint is thus a mendelian separation based upon a number of factors.[1] Similar results occur in crosses between Europeans and Hottentot women. Here too we get a sorting out into various tints. Among these, persons of a comparatively pale colour are more numerous, so that some factor of paleness must be dominant. The remarkable fact that persons of mixed Tamil and Malay stock are often darker than either parent, and the fact that persons of mixed Portuguese and Indian descent, when they interbreed, sometimes produce offspring of an almost jet-black tint, are explicable through a study of the result of similar crossings made by animal breeders (polymery).

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[1] Davenport, Heredity of Skin Colour in Negro-White Crosses, "Carnegie Publications", no. 188, Washington, 1913.

As regards the crossing of blonds and brunettes in Europe, further investigation is needed to elucidate many of the details. Here likewise, however, there is a manifest tendency to the separating out of the various tints, and we have to assume that a number of heredity factors are at work. The only suitable persons for study are those who sunburn permanently and strongly. We find that there are individuals who burn intensively and equably; others who only become brown after a stage of redness and irritability of the skin; others who bronze very little in the sun; and yet others in whom repeated exposure repeatedly induces redness, inflammation, and the formation of bullae. There will be found (even among blonds) more persons who brown under the sun than is usually supposed if data are secured only by the examination of persons who have been little exposed to sunlight.

7. COLOUR OF HAIR

The different tints of the human hair are due, not only to differences in pigment, but also to differences in structural conditions. As far as pigment in the hair is concerned, it exists both in pigment grains and diffusely. The pigment grains are situated in the cortical cells, and also between these, sometimes only in a few cells (fair hair), sometimes in thick masses in all the cells (dark hair). The medullary cells can also contain pigment granules. Diffuse pigment has been shown unquestionably to exist in red pubic hair, and in the yellowish-blonde hair of partial albinos. Perhaps it is always present in red hair.

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[1] See above, pp. 64–5.

Of importance to the colour of the hair is its air content and its greasiness (sebaceous glands). Sun and sea-water have a bleaching effect, and the possibility of such paratypical influences must always be taken into account.

In old age, as every one knows, the hair pigment disappears. The hair becomes first grey, and then white, the change being jointly due to the appearance of air-bubbles in the hair and to the disappearance of pigment. Senile whitening of the hair sets in at varying ages, and it is a familiar experience that early or late whitening of the hair tends to run in families; but further careful study of the fact is desirable.

All these factors in their manifold combinations result in the production of a very large number of different tints of hair in the human race considered as a whole. From the fairest yellowish-white and the most delicate silver-grey, the colour scale runs in two parallel series to black, the former by way of gold-blond, gold-brown, and brown, to the deepest black-brown, and the latter by way of silver-grey, ashen-grey, and dark grey (to be distinguished from the grey of old age), to a true black. Hair belonging to the first series has always a reddish element in its tint. We may to-day take it as certain that these differences in the colour of the hair are racial.[1] (See below, p. 188.) In birds and mammals it has been shown that in the climatically determined development of local races the various melanin pigments react very differently to northern climates, damp and warm climates, etc.[2]

A large majority of all the races of man have very dark black-brown or black hair. Light brown and blond hair as a racial characteristic was originally known only in Europe.

Inheritance of the colour of the hair displays mendelian separation. Even in the absence of numerical proof of this, the following considerations would be convincing. For more than a thousand years, fair-haired and dark-haired types have been mingling in Europe, and there has not yet resulted the formation of a uniformly intermediate tint of medium brown or light brown, for we continue to see persons with dark-brown hair side by side with those of exceedingly fair hair. For countless generations, black-haired and red-haired Jews have been interbreeding, and they have not produced an equably dark reddish-brown stock. Furthermore, when we make a statistical study of particular cases, we find that the children of a marriage between persons having differently coloured hair do not themselves possess hair of a uniform tint midway between that of the two parents, but that the colour of the hair is very variously inherited. This manifest alternation in the inheritance, this unmistakable failure to form an intermediate hereditary tint, is a very striking proof of the constancy of racial characteristics, of their persistence through innumerable generations, of the possibility of cultivating or eliminating such characters by purposive breeding.

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[1] Late darkening (see below) seems to run a different course in hair of the two different colour types. (Information communicated by letter by J. Czekanowski).

[2] Haecker, Klima und tierische Pigmentierung (in the press).

The observation of abundant statistical material, studied in detail, shows that this mendelian separation occurs. Speaking generally, children are not darker-haired than the darker-haired of their parents. Occasionally, however, we find that they are, and this can be explained by the working of factor combinations (see pp. 55 et seq). In like manner, the crossing of a blond European with a light-haired albino Negro produces a typically dark mulatto. Conversely, the tint of hair in the children is often lighter than that of the lighter-haired of the two parents (in whom there is a masked, still lighter strain). The darker tint masks the lighter, fair hair being recessive to dark.

What has here been established in the case of European children, was also found to apply to crosses between Europeans and Negroes, and between Europeans and Hottentots.

We must, therefore, assume that the hereditary transmission of the granular pigment of the hair is dependent upon several factors, this being accordant with our experience in the case of the lower animals (rabbits, mice, etc.). There appears to be one basic factor for pigment formation, *A*, so that *aa* individuals have albinotic white hair (see below). Then there are several (*Z*, *Y*, *X*, . . .) factors for the formation of brown pigment, each of them rendering darker the colour produced by the one before. Parallel with these (perhaps corresponding to the "grey" series of hair-tints) there is a series of factors (*K*, *J*, *H*, . . .) which produce a similar effect in hair belonging to this series. At work in the first series, also, there is a red factor, or perhaps more than one red factor. We have to assume that it exists side by side with the brown factors as a separate factor, which, without notably altering the intensity of the brown tint, is superadded to brownness to produce a redness. We will call this factor (or these factors) which perhaps produces a diffuse pigment, *R*. Red is then dominant over not-red. Individuals belonging to the "grey" series are all *rr* persons. In the case of hair of the "brown" series ranging from very fair to medium brown, the presence or lack of the red factor to a varying extent induces a varying degree of redness; but in the case of the darker grades of brown hair. the red colour, if present, is masked.

The working of these various heredity factors will account for the varying frequency in the occurrence of red-haired persons. We do not know, however, whether the occurrence of isolated groups in which redness of the hair is especially frequent and conspicuous (as among the Melanesians of New Guinea) is due to a spontaneous appearance of the red heredity factor, or to the disappearance of a factor which has hitherto masked the redness or rendered the red factor inoperative. The red factor is plainly at work in connexion with the appearance of freckles.[1]

Similar considerations apply to the appearance of blonds in a widely distributed race like the Nordic. Besides, this is the only race which we know to be genuinely blond. (See below, p. 186.)

In Baden, Denmark, and Scotland, and in the mixed breeds of South West Africa, it has been shown that the women have darker hair than the men. This, doubtless, must depend upon the localisation of certain pigment-forming factors in the sex chromosome.[2] Since the human female is in this respect homozygotic, more female individuals must receive this dominant pigment factor than males. It will follow, therefore, that the males will more often inherit the pigment factor from the mother than from the father. That is in conformity with a widespread popular opinion to the effect that dark-haired fathers and blond mothers more often give birth to blond sons and brunette daughters, than conversely. The notion, however, requires statistical confirmation.

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[1] Siemens, "Arch. Dermat.", vol. CXLVII, 1924.

[2] Lenz, Ueber die krankhaften Erbanlagen des Mannes, Jena, 1912.

In certain cases, at any rate, like considerations apply to the brown tint of the eye. In Swedes, Finns, and Lapps, brown eyes are so much more frequent in women than in men, that we are forced to assume the existence of a sex-linked heredity factor.[1]

A remarkable phenomenon in the colour of the human hair is the so-called late darkening. Every one knows that a great many blond children acquire dark hair as they grow up. Careful study has shown that in various black-haired races, such as the Japanese, the South American Indians, and perhaps the Papuans (Negroes have not been studied), little children have their hair a shade lighter than adults. To the hair, then, the same thing applies as to the skin—pigmentation grows darker during the years of childhood. But this is not the same as what we know as late darkening in Central Europe. Here a great many children are pale blond, gold blond, reddish blond, or sometimes yellowish blond—and when grown up the same individuals are brown, dark brown, and even black brown, in the tint of their hair. Among the mixed breeds in South West Africa, the offspring of blond Boers and dark-haired Hottentot women are all blond in youth (many among them having at the same time the frizzled hair characteristic of the Hottentots), whereas the adults usually have dark and even black hair. This very remarkable change in the tint of the hair must be regarded as an outcome of hybridisation. The ancestry of the persons concerned certainly contained both blonds and brunettes.

The formation of the hair pigment appears to be determined by hormones, whose working is renewed and intensified at puberty. As already said, the hereditary transmission of the colour of the hair depends upon numerous cooperating, mutually reinforcing, heredity factors. Lenz writes: "The degree of late darkening under the influence of the hormones will therefore depend upon the kind and number of extant pigment-factors."[2] In blonds of pure race these pigment factors are so few in number, and in brunettes of pure race they are so numerous, that the hormonal effect is but slightly manifest. In persons of mixed stock, however, who have diversified pigment-factors present in varying numbers, the intensified hormonal action at puberty is capable of bringing about a marked late darkening. The fact that in some of those who are born with dark hair the hair subsequently becomes light, seems also to be due to hormonal influence (perhaps a result of the maternal hormones upon the embryo, or perhaps a result of the hormonal influences being stronger in the embryo than in the suckling—compare the mammary development of the embryo and of the suckling).

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[1] Lenz, Ueber geschlechtsgebundene Erbanlagen für Augenfarbe, "Arch. Rass. Ges. Biol.", vol. XIII, 1921.

[2] "Arch. Rassen und Ges. Biol.", vol. XVI, 1925.

Finally we have to consider a lack of pigment in the hair. In partial albinism we may find isolated bundles of white hairs. This is often associated with the appearance of albinotic patches in the skin. The phenomenon is at least as common in the coloured races as in the white, and in the former it is naturally far more striking. Albinism is a dominant hereditary character. It must, therefore, be regarded as an inhibitive factor, causing a loss of pigment in various parts of the skin. Numerous genealogical trees have been recorded showing the recurrence of this phenomenon in several generations. In contrast with this we have total albinism. Here the body forms no pigment at all. The hair is white, the skin is dead white, the iris is pigmentless and of a red tint from the blood shining through. This lack of pigment is an idiovariant by defect, the basic factor *A* for producing pigment is wanting. Total albinism, therefore, is recessive. We have a number of genealogical trees. The phenomenon is quite as common in the coloured races as in the white races. Very frequently, however, the albinism is incomplete, or the condition becomes somewhat less marked in the adult. In such cases the "white" hair is of a pale yellow, and the skin (in albino Negroes) is of a dirty white or yellowish white. One or other of the factors of pigment formation is present, and takes effect after a time, but, being alone, too late.

Let us now consider the occurrence of idiovariations and mixovariations, that is to say the hereditary transmission of subordinate phenomena that are due to peristasis. Do paravariations occur in this domain?

Certainly there are no marked and plain paravariations.

I may mention in this connexion the bleaching of the hair by the sun and by salt water, which makes it difficult to determine the true tint of the hair according to the scale; and moreover, the effects of like exposure vary much from individual to individual.[1] For the rest, we can nowhere find any evidence of an effect of climatic factors in the way of directly changing the colour of the hair, nor is any such change of colour produced by nutritive or other influences. (Apart from the whitening of the hair due to old age, and from the sudden whitening which sometimes results from fright, or shock.) The distribution of the colour of the hair throughout the world does not correspond in any way to climatic zones. To sum up, the colour of the hair would therefore seem to be a markedly racial character.

8. TINT OF EYES

In mammals, the iris owes its colour to a two-fold pigmentation. The posterior stratum of the iris is covered with a double layer of deeply coloured epithelial cells, which alone suffice to render the membrane opaque, and to prevent the entrance of light-rays into the interior of the eye. In front of this there are interspersed throughout the tissue of the iris a number of larger and smaller branched pigment cells. There is, however, one exception to this structure common to all mammals. In some of the fair races of various domesticated animals, pigment cells are completely lacking in the anterior tissues of the iris, which then appears light yellowish brown, or grey, or green, or blue.[2] (Blue when the pigment cells are completely absent. Then the constantly present pigmented iris epithelium shines through the semi-transparent anterior layers of the iris and this gives a blue tint.) In human beings as in most other mammals the colour of the eyes is generally dark, but sometimes, as a racial character, in the human species as in certain exceptional stocks of domesticated animals, the pigment in the anterior layers of the iris may be wanting.[1] In this way we get light brown, grey, green, and blue eyes, as a racial character. The same lack of pigment in the anterior layers of the iris occurs also in individual instances, in cases of partial albinism in members of a normally dark-eyed race. For instance, we see this sometimes in Negroes, who then have blue eyes, a fair skin, and yellowish-white hair. In cases of complete albinism, the absence of pigment may go a stage further, affecting also the epithelium on the posterior surface of the iris. Then the bloodvessels show through the transparent membrane, and the eye is red or pink.

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[1] Siemens, "Arch. für Dermat.", vol. CXLVII, 1924.

[2] Hauschild, Untersuchungen über die Pigmentation im Auge verschiedener Menschenrassen, "Zeitschr. Morph. Anthr.", vol. XII, 1909.

We are entitled to assume that the absence of pigment in the anterior layers of the iris is the outcome of a variation by defect. It follows that the hereditary transmission of a light colour of the eyes is to be regarded as a recessive mendelising character. Observation confirms this inference, and shows that the loss of (at least) one heredity factor results in the eyes being light-brown or grey, and that the loss of an additional heredity factor results in the eyes being blue. Parents with pure blue eyes have blue-eyed children. In heterozygotics, the greyness, greenness, or blueness of eyes which would otherwise be grey, green, or blue, may be masked by the presence of brown pigment; and the children of parents having brown eyes of this kind may have grey, green, or blue eyes. Parents with dark brown eyes will either have children whose eyes are also dark brown, this happening when both parents are themselves homozygotic (as is the case in all the dark-skinned races); or, if one of the parents is homozygotic while the other is heterozygotic, all the children will still have dark brown eyes; but when both parents are heterozygotic, some of the children will have light-coloured eyes.

In exceptional cases, however, even when the parents have pure blue eyes there would seem to be a heterozygotic element at work, leading to the presence of an additional factor which prevents the "brown" making its appearance. (Usually in such instances we note other "defects" in the eyes, such as anomalies of refraction.) In these circumstances, of course, a brown-eyed person will turn up here and there among the offspring.[1]

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[1] E. Fischer, Die Rassenmerkmale des Menschen als Domestikationserscheinungen, ibid., vol. XVIII, 1914.

The fact that the colour of the eyes is not always fully developed until some months after birth (perhaps this also happens occasionally in the coloured races), and that light-coloured eyes may even become dark much later than this, at the age of ten to twelve, is doubtless connected with the above-described process of pigment formation. Peculiarities of the same process explain the fact that in advanced age the eyes tend to become lighter. In brown eyes, a moderate decolorisation occurs; in blue eyes, when the tissues of the iris become denser in old age, and therefore less transparent, the pigmentary layer does not show through so well, and the thickened connective tissue acquires a pale, watery-white appearance.

What is called the white of the eye is only white in Europeans; in the coloured races it is of a dirty greyish-yellow colour, marbled with light brownish-yellow. In this region, the deeper cell layers of the conjunctival epithelium are always pigmented (as in all mammals, with the exception of the very light-coloured races of domesticated animals).

The influence of external factors upon the colour of the eyes can apparently be excluded, so that here we have to do with unmistakably racial characters.

In conclusion, I must refer to what is termed the complexion. By this is meant the general tint of skin, hair, and iris. In the great majority of persons, even in those of mixed race, the relative degree of pigmentation is identical in all three of these. We must suppose, therefore, that the same heredity factors condition the pigmentation of all three of these parts simultaneously. Still, the exceptional occurrence of disharmonious tinting, shows that there must be factors which influence the pigmentation of the three parts separately. When this occurs, a disharmony between the colour of the hair and the colour of the iris is much rarer than a disharmony between the colour of the hair and the colour of the skin. Among the Germans, blonds with a skin which is capable of becoming equably bronzed (and must therefore be richly pigmented) are not uncommon.

Little attention has hitherto been paid to the fact that in Germany among black-haired (and black-eyed) Jews of the Sephardic type the skin is almost invariably poor in pigment and peculiarly white. (I do not know if the same thing is found in Southern Europe and Northern Africa.)

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[1] Winge, "Zeitschr. für indukt. Abstd.", vol. XXVIII.

In like manner, the difference in tint which is frequently noticed between the hair of the head and the hair of other parts of the body (including the pubic hair and the hair of the beard, which go together), shows that individual factors for the pigmentation of the hair of these different regions must be taken into account in addition to the general factors for the pigmentation of the hair. Here in Germany, we often see men who have brown hair on the head but fair or reddish hair elsewhere on the body, whereas the reverse is hardly ever seen. When the hair of the head is fair, the hair of the body is also fair or red. In blond women, on the other hand, the pubic hair is usually of a darker tint than the hair of the head.

9. GROWTH OF HAIR

Whereas in nearly all mammals living in a state of nature we find a practically uniform distribution of the hairy covering of the body, smooth and straight or woolly as the case may be (all members of the same species having the same sort of hairy covering), among human beings there are well-marked racial differences in the growth of the hair. But among domesticated animals we find, as we find in man, great differences in this respect within the same species (a woolly coat in some animals and an Angora coat in others).

The fact that in frizzy-haired New Caledonians, just as among Negroes, the children have straight or wavy hair (as well as comparatively light hair), shows that in the primal human type the hair was straight, just as it is in all the monkey kind.[1] Such straight hair, hanging limply and smoothly, closely applied to the surface of the body, and flowing down softly from the head, is typical of Europeans, Australian Blackfellows, the aboriginal Veddahs of Ceylon and the Senoi of Malacca, and the Ainu of the northern island of Japan. When the hair is in other respects of this type but has become thicker and harsher, it is "stiff" as in Mongoloids, Indians, and Eskimos. If, on the other hand, the slight curl sometimes seen in "straight" hair, becomes intensified, we get "wavy" hair, in which the waves may be either broad or narrow. Finally, if the hair is separated into locks by spiral twisting, we get the typical

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[1] F. Sarasin, Anthropologie der Neukaledonier, etc., Berlin, 1916–1922.

"frizzed" or "kinky" hair, in which the spirals of hair combine to form a thick woolly coat, as in many of the Negro races and in the Melanesians. There is a more extreme type of this woolly hair in which the spirals are very small and closely set, the hair being short, and adjoining spirals being interwoven to form little clumps; of this kind is the hair in Bushmen, which is sometimes spoken of as "fil-fil" or "peppercorn hair".

Since in Negro children the hair assumes the adult type of growth and the adult black colour much sooner than in the New Caledonians, we infer that the frizziness of the hair in these two groups does not denote any close kinship between them (Sarasin). It seems to me that in the two cases the character must have originated independently as a phenomenon of domestication.

From crossings between straight-haired and curly-haired Europeans, as well as from crossings between Europeans and Hottentots and between Europeans and Negroes, we learn that if both parents are straight-haired all the offspring will be straight-haired. We may assume the straight-hairedness to be a foundation to which the factor *C* "producing curliness" can be superadded. Thus straight-haired people are *cc*. Parents with loose wavy hair, parents with curly hair, and parents with very closely set curls, have children with all these different growths of hair. Loosely-waved-haired and curly-haired people are heterozygotes (*Cc*). We are probably entitled to assume that hair with a really spiral twist is produced by a second factor. The before-mentioned *C* makes a bend or a wave, whereas the factor we are now considering, *S*, transforms this into a spiral twist. Very closely curled hair must, therefore, be *CC SS*, whereas straight hair must be *cc ss*.[1] Probably *S* cannot operate unless *C* is present; this may explain why, among the Jews for instance, closely curled hair will appear as a seeming "sport", apparently uninherited.

A study of the offspring of crossings between Chinese and Filipinos,[2] tends to show that the stiff hair in such hybrids is dominant, differing in this respect from the straight hair of Europeans. We must assume, then, that a "factor tending to make the hair stiff" is at work—a factor lacking in the comparatively fine straight hair (and the curly hair) of Europeans. We may term this factor *L* (lissotrich).[1]

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[1] Fischer, Rehobother Bastards (see full reference in bibliography).

[2] Bean, Heredity of Hair Form among the Filipinos, "Amer. Natural.", vol. XLV, 1911.

Turning to consider the general distribution of the hair and the quantity of hair on other parts of the body than the scalp, we find that in this there are marked racial differences. The extent of hair on the hairy scalp is, however, much the same in all races.

The whorl over the junction of the two parieto-occipital sutures is (in the population of North Germany) clockwise in 74 %, and counter-clockwise in 19 %; in 7 % there is a double whorl. Very rarely do we find three whorls or more. The sexes are alike in this respect. Inheritance of this character is manifest, the clockwise direction of the whorl being dominant. (There seems to be a pair of heredity factors at work.) Observations on other races are lacking. (Bernstein, Sitzungsber. preuss. Akad. der Wissensch. Phys. Math. Kl., 1925.)

Marked hairiness of the body and limbs, which is fairly common among our own people of northern Europe and the Mediterranean region, is found to an even greater extent in some of the groups of Asia Minor, in the "hairy Ainu", and in the Australian Blackfellows. All other races than those named, on the other hand, are less hairy than Nordics and members of the Mediterranean races. Least hairy of all are the American Indians and the Eskimos.

The geographical distribution of these phenomena indicates that they are not the outcome of external influences, but must be hereditary racial characters. The same is true of the abundance and the shape of the beard. In the Japanese, the beard appears later in life than among Europeans; but in Japanese, Negroes, and Melanesians, it has the same shape as with us. On the other hand, in the Veddahs, the beard covers only the lower surface and the point of the chin, being shaped like a goat's beard, while whiskers and moustache are wanting or almost entirely so. In this case, likewise, we obviously have to do with hereditary racial factors. The same thing applies to such a character as the coalescence of the eyebrows above the nose, which occasionally occurs among our own people, but is common in Asia Minor and in Crete.[1]

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[1] In this connexion there is urgent need for a study of Eurasians of mixed European and Mongol descent, and this study would throw much light on other domains of anthropology as well.—Schlaginhaufen has recorded the appearance as a variation of very closely curled hair in a boy in Switzerland ("Verh. Schweiz. Nat. Ges.", 1922). We must expect on theoretical grounds that such "idiovariations" will occur from time to time.

10. SKIN

In many parts of the surface of the skin we find folds and wrinkles, and in some parts much deeper furrows. The folds and wrinkles are the expression of the frequent flexing of the skin as the outcome of regular muscular activities. The flexion-folds of the anterior surface of the hands and the fingers (the so-called "M-figure") are already produced before birth by the movements of the embryo. The marked individual differences in the distribution of these folds are determined by innumerable slight differences in the arrangement of the muscles, the position of the joints, the distribution of the fatty layer beneath the skin, and so on. Speaking generally, these characters are determined by inheritance, so that the flexion-folds of the skin are hereditarily transmissible.[2] The same thing applies to the folds in the face, but here detailed investigations are still lacking. Quite different from these folds are the ridges and furrows which form fine patterns of lines (the papillary ridges) on the palmar surface of the hands and fingers and the plantar surface of the feet and toes. On the finger-tips, the figures made by these ridges are extremely complicated, being groups of arches or spirals or double spirals intersecting one another in various ways. Those on the feet are essentially like those on the hands, but much less strongly developed.

The arrangement of these furrows is a purely individual character, no two persons being found alike in this respect among many millions. Thus has arisen the science of dactyloscopy, the scientific study of the arrangement of the "lines" on the finger-tips, which can be classified for the purpose of the identification of criminals, and therefore has become of great social importance.

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[1] How complicated the conditions of hereditary transmission in these matters must be, however, is shown by the fact that in European males part of the hair of the face, namely the central part of the moustache and the upper part of the whiskers, is influenced in its growth by the testicular hormones; whereas the beard proper and the lateral parts of the moustache are influenced by the pituitary hormones. (Olivet, "Zeitschr. Konstit.", X, 1924.)

[2] Hella Pöch, Ueber Handlinien, "Mitt. Anthr. Ges.", Wien, 1925, p. 133.

But apart from these individual differences, particular kinds of patterns, of spirals, arches, etc., form a racial character. Thus, there are certain details of resemblance between Japanese, Koreans, and Chinese, which show their interconnexion, whereas the patterns in the Ainu are very different. The Anglo-Americans and the Hindus differ markedly from the aforesaid inhabitants of eastern Asia; and the Negroes, again, differ from both these groups. A detailed study of this subject would be of great importance. In especial, it might throw considerable light upon the racial interconnexions of Europeans.[1]

The arrangement of the papillary ridges is controlled by definite heredity factors, but the relations seem to be exceedingly complicated. At least five similar and mutually reinforcing factors appear to be at work.[2] Mon-oval twins often exhibit an extraordinarily similar arrangement of the ridges; but sometimes we note considerable differences in these cases, just as there often are considerable differences in human beings between the arrangement of the ridges on the right hand and on the left. Nevertheless, in such mon-oval twins there is always a much closer resemblance between the arrangement of the ridges in the two (especially when we compare the same hand and finger in both) than there is between ordinary brothers and sisters, so that a comparison of the finger-prints gives us one of the best means for convincing ourselves that twins are mon-oval.[3] Let me emphasise the fact that, once the ridges have appeared in early intra-uterine life, there is no subsequent change in their number and arrangement.

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[1] Poll, Ueber Zwillingsforschung, etc., "Zeitschr. Ethn.", 1914. (Contains bibliographical information.)—Hasebe, Ueber das Hautleistensystem der Vola und Planta der Japaner und Aino, "Arb. Anat. Inst. Sendai", 1918.—Kubo, Beiträge zur Daktyloskopie der Koreaner, "Mitteil. Med. Keijo.", 1918. H. H. Wilder, Racial Differences in Palm and Sole Configuration, "Am. Journ. Anthr.", vol. V, 1922.—H. H. Wilder, "Biol. Bull.", vol. XLIX, 1925.—Miyake, Ueber das Hautleistensystem der Vola der Koreaner, "Zeitschr. Morph. Anthr.", vol. XXV, 1925.

[2] Bonnevie, Zur Analyse der Vererbungsfaktoren der Papillarmuster, "Hereditas", IV, 1923.

[3] Ganther and Rominger, Ueber die Bedeutung des Handleistenbildes für die Zwillingsforschung. "Zeitschr. Kinderheilkunde", vol. XXXVI, 1923, p. 212.

Among Europeans there are great individual differences in the thickness of the skin. As regards this considered as a racial character, we know very little, but it appears that the skin of the desert folk of North Africa is exceptionally thin and poor in fat. Hottentots and Bushmen show a marked tendency to the formation of wrinkles in the skin. There are also differences in the number and the arrangement of the elastic fibres in the skin of the face.

In Mongoloids, there is not so marked an accumulation of sweat-glands in the armpits as with us. Speaking generally, the glands of the skin exhibit certain racial differences, their minute structure varying as between Australian Blackfellows, Chinese, Cameroon Negroes, and Europeans.[1]

11. STATURE

A comparison of human beings in respect of stature discloses, not merely marked individual differences, but also a considerable variation in the mean stature of different groups. Careful investigation shows that the conditions are extremely complicated, being dependent upon an association of idiotypical and paratypical phenomena. In all human beings, the processes of growth normally run much the same course. Even the sexual differences in the curve of growth, the surpassing of boys by girls before puberty, and the subsequent overtaking and surpassing of the girls by the boys, are alike in all races. In the races whose mean stature is small, growth is usually arrested several years earlier than in the tall races, but the members of the former are also somewhat smaller at birth.

Within any one race, we sometimes find that exceptionally tall persons have had an above-average length of body from the very first; in other cases, exceptionally tall persons have only begun to become so rather suddenly, at puberty. This circumstance alone suffices to show how complicated the conditions are.

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[1] Schiefferdecker, Die Hautdrüsen des Menschen und der Säugetiere, "Zoologica", no. 72, Stuttgart, 1922.

Growth in stature is dependent upon the internal secretions. The thymus (?), the pituitary, and the gonads or reproductive glands, influence the stature. If the functioning of these glands is disordered, the result is the production of a giant or of a dwarf, as the case may be. The relative proportions of different parts of the body and limbs are likewise affected in this way, as can be seen from a glance at Fig. 27, facing p. 152, depicting a man in whom an under-development of the testicles has resulted in growth to a height of 6 feet 2 inches, with an extremely disproportionate length of the limbs resembling that common in eunuchs. We do not know whether such glandular anomalies are hereditary, but it seems not unlikely that they are.

The stature is notably influenced by general nutritive conditions, as well as by the internal secretions. Experiments on animals show that dogs, or pigs, of the same litter can be made to grow to very unequal sizes by differences in their respective food supply (see above, p. 30). The same thing applies to the human species. The stature, the chest measurement, and the circumference of the head are, on the average, notably smaller in children of the poorer strata of the population than in children of the same age among the well-to-do.[1] Probably these factors account for the marked increase in stature of the European population during the last hundred years. In Scandinavia, the average stature of recruits has increased from 1½ to 2 centimetres during the last twenty to thirty years; in Baden during forty years it has increased by 1·2 centimetres; and among the Dutch Jews there has been an even greater increase.[2]

By extraordinarily careful study, Martin has been able to show that the under-nutrition of the German youth during the war and post-war periods has notably reduced their stature, their body-weight, and their chest measurement. Of late an increase towards the earlier recorded dimensions has been demonstrable.[1]

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[1] We must remember the possibility of racial differences as between the upper class and the lower. Cf. Niceforo, Anthropologie der nichtbesitzenden Klassen (a translation from the Italian), Leipzig, 1910. (There is also an abbreviated French version of Niceforo's book, entitled Les Classes Pauvres.) But nutritive conditions have more effect upon these characters than has racial inheritance. See Herskovits, "Amer. Journ. Phys. Anthr.", 1924.

[2] Bolk, De lichaamslengte van Amsterdamsche Joden in 1850 en 1900, etc., "Nederl. Tydschr. Geneesk.", 1910. (See also "Zeitschr. Morph. Anthr.", XVIII, 1914.)

The foregoing are parakinetic influences. Now the idiogenic influences must be considered. It has long been a matter of common observation that the members of some families tend to be tall and those of other families to be short. King Frederic William I of Prussia would only give his tall grenadiers permission to marry if they chose brides who were likewise of great stature, for he wished to breed a race of tall soldiers. The recent study of racial crossings among human beings has gone far to show that the inheritance of stature must depend upon dominant mendelising factors, and that probably we have to do here with polymeric inheritance. One notable fact observed in such racial crossings, a peculiarity with which the hybridisation of animals and plants has already made us familiar, is a tendency on the part of cross-breeds to be larger and stronger than either parental race.

In human beings, the cross-breeds derived from tall North American Indians and tall members of the white race, living under the same conditions as their parents, are taller than either of these. In like manner, cross-breeds between Boers and Hottentots are on the average taller than the members of either parental race.[2] Even the mingling of types much more closely akin than the foregoing seems to increase the stature, as has been shown by certain observations made in Norway,[3] and also on Negro-Hamitic crossings.[4]

There has been much discussion concerning the origin of the so-called Pygmies, or dwarf-races. They are certainly not the outcome of a dwarfing due to unfavourable nutritive conditions; they are idiovariations, in whose origin and maintenance selection must unquestionably have played a part. The separate Pygmy races

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[1] Martin, Die Körperentwicklung der Münchner Volksschulkinder, etc., "Anthr. Anz.", I, 1924, p. 76.

[2] Boas and Fischer, op. cit.

[3] Waaler, Heterozygoti og legemshoide, "Arch. Math. Nat.", vol. XXXVIII, no. 6, 1923.

[4] Struck, Versuch einer Karte des Kopfindex im mittleren Afrika, "Zeitschr. Ethnol.", vol. LIV, 1922, p. 78.

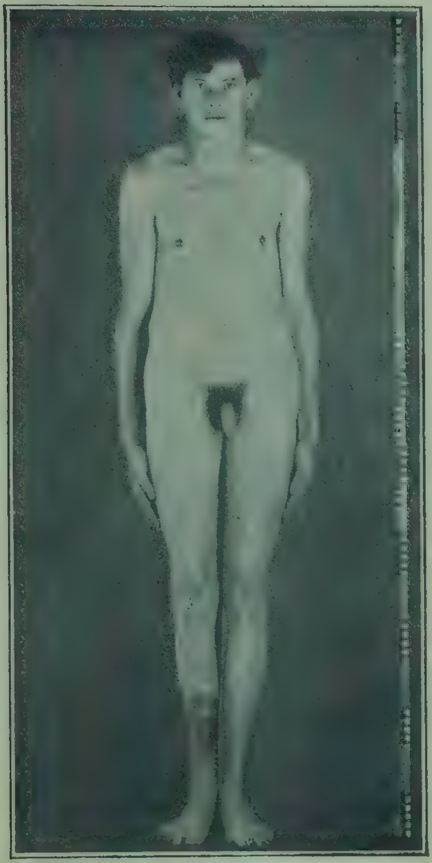

FIG. 27.

Excessive stature due to under-development of the testicles. (After Henckel.)

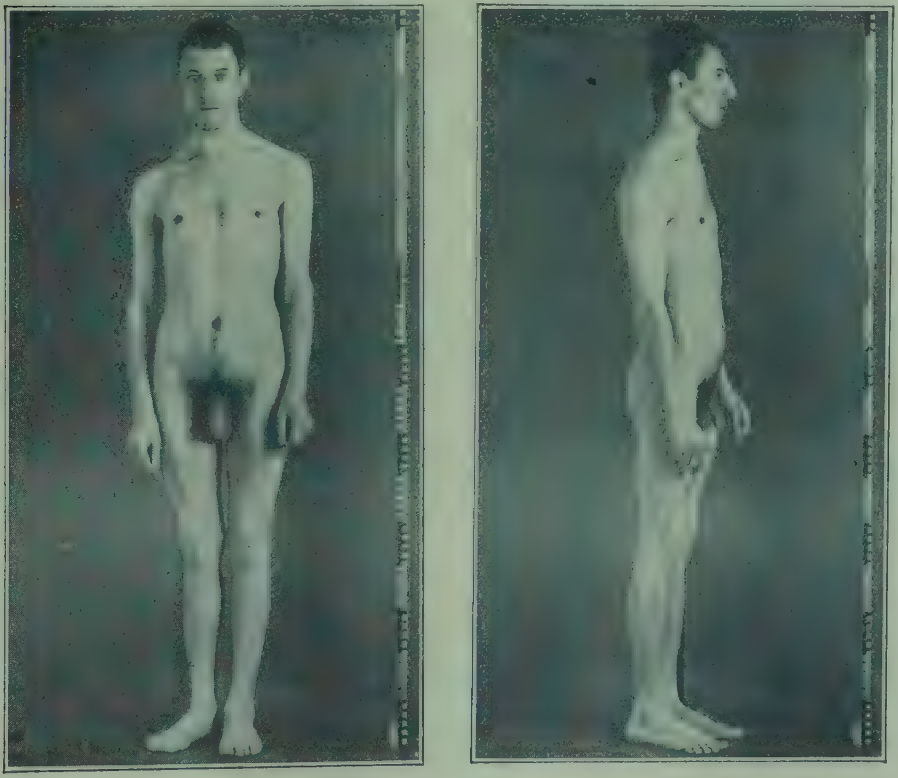

FIG. 28.—Asthenic type.

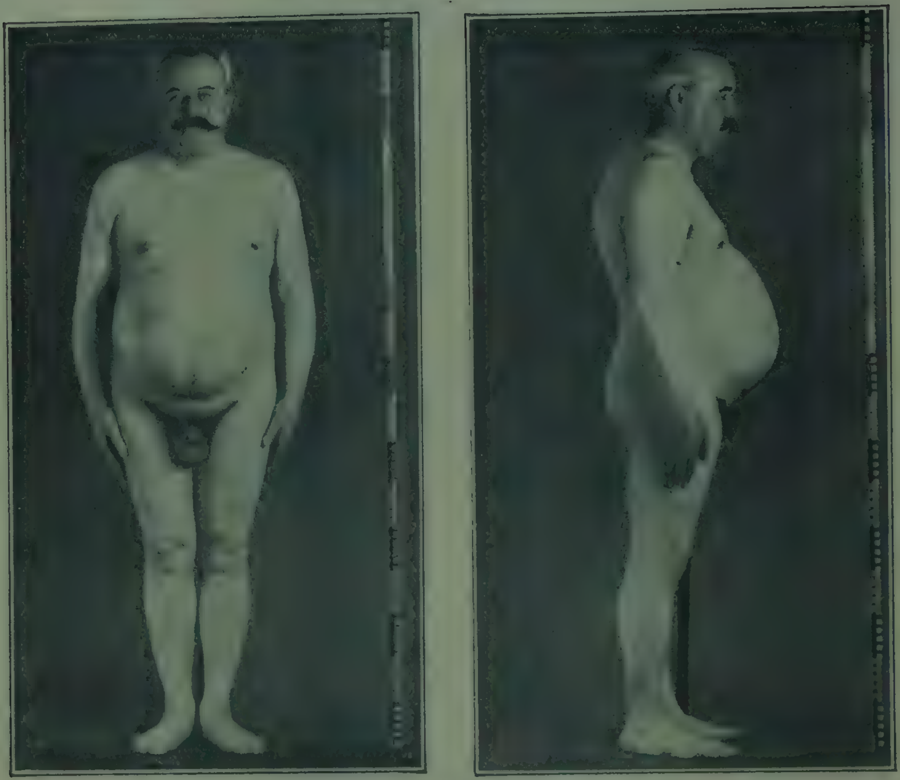

FIG. 29.—Pyknic type.

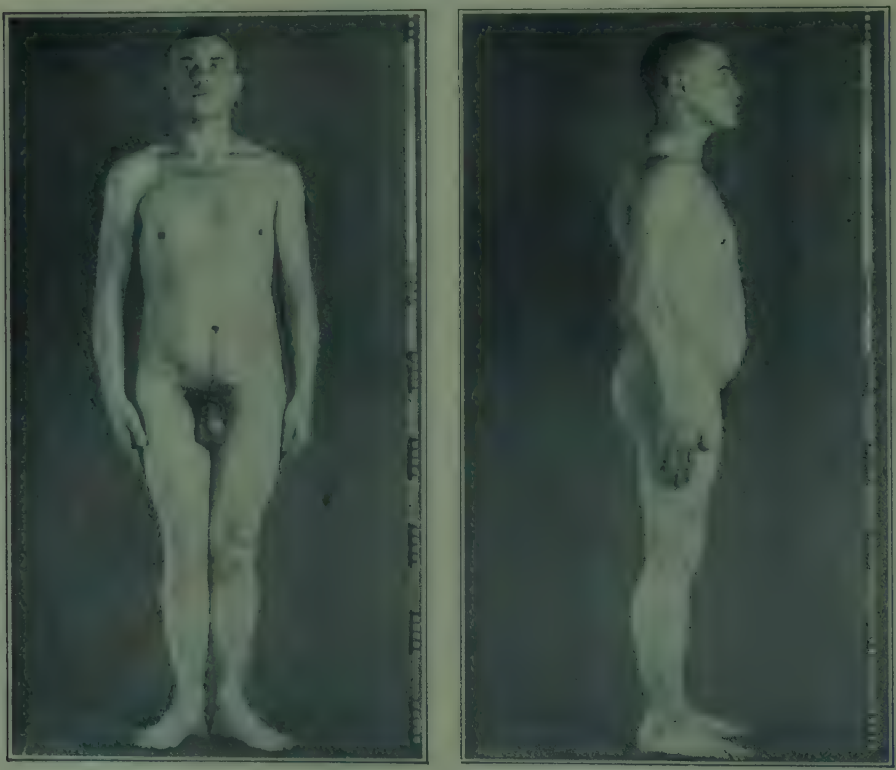

FIG. 30.—Athletic type.

must have independently split off from other races. A study of "racial stature" shows plainly how the hereditary type of growth characteristic of the race persists amid all the modifications due to the working of external influences upon the individual, and however much in individual instances it may be masked by paratypical modifications.

12. PROPORTIONS AND SHAPE

The proportions of the body, that is to say the natural ratios between the different parts of the body in respect of length and size, and their ratios to the body as a whole, are so largely dependent upon the effects of external influences that such racial distinctions as may exist in these matters are for the most part masked. Nevertheless, the comparative length of the legs in Negroes, and similar characters in other races, are manifestly hereditary, this being proved by the unmistakable predominance of shortness of the limbs and narrowness of the hips in members of the first generation of crosses between long-limbed Australians and Europeans of short stature—but the details of these matters need further investigation.[1] Independently of racial differences, the women of all races have, as compared with the men, shorter legs and a longer trunk.

A paratypical effect is the greater length of the arms in such persons as blacksmiths whose occupation involves a preponderant use of the arms, whereas students, officials, etc., have relatively longer legs, but seafaring men relatively longer arms and legs. Thus the use of the extremities tends to increase their growth in length. Speaking generally we may say that gymnasts, heavy athletes, runners, etc., have their respective peculiarities in chest measurement, stature, and so on.[2] We have to remember, however, that many of the peculiarities that come under this head may, as far as essentials are concerned, have been inherited, and may have induced the person concerned to adopt a particular occupation or to practise a particular form of sport. It remains uncertain, also, how far social selection is operative in these matters. The mingling of races in the European population would seem competent to make it peculiarly effective.

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[1] See Davenport, Inheritance of Stature, "Genetics", II, 1917.

[2] Martin, Die Körperbeschaffenheit der deutschen Turner, "Monatsschr. ür Turnen, Spiel und Sport", vol. III, Berlin, 1924.—Bach, "Anthr. Anz.", 1924, p. 200, and 1925, p. 167.

This is the appropriate place for the consideration of right-handedness. We know it to be the rule among human beings. Still, right-handedness gives another indication of the immense difficulties that assail us when we strive to distinguish between hereditary foundations and parakinetic influences. Nothing is really known about the origination of right-handedness or left-handedness. Unquestionably, however, in many individuals there must be an anatomical basis for the preference given to one hand over the other. This is shown beyond question by the fact that so many left-handed persons remain left-handed in defiance of the most strenuous attempts to educate them to right-handedness.

The left-brainedness or right-brainedness which underlies "in-born" right-handedness or left-handedness, depends either on directly operative heredity factors, or else upon non-hereditary conditions concerning the position, the flexure, the vascular ramification, etc., of the embryo.

No trustworthy statistical inquiry has yet been made to throw light upon the hereditary transmission of left-handedness. The results of the investigation of twins do not support the hypothesis of inheritance.[1]

A habitual predominance of the left leg over the right is far less marked than the habitual predominance of the right hand over the left [2]—doubtless because the functions of the two legs are far less differentiated than are the functions of the two hands, and because where they are differentiated this has been mainly due to education.

The whole body is moderately asymmetrical. Most people have a slight curvature of the spine, with a convexity to the left in the dorsal portion and to the right in the lumbar portion; but in the minority the curves go the other way. In this matter, likewise, the position of the embryo and the conditions of intra-uterine growth are the probable determinants.

Very numerous factors combine in an extremely complicated way to determine the general form of the body, whether it is thin or fat, slender or thick. Nutritive conditions play a great part here, as was plainly shown in Germany by the considerable changes in the habit of body which occurred during the war period and the years of inflation. But there is also evidence to show that marked hereditary differences exist in the individual type of nutrition and in the way in which the individual reacts to different quantities and kinds of food, so that, under identical conditions, different individuals may deposit very different quantities of fat. Whole groups and races of man show hereditary differences in these matters. For instance, North African Jewesses have a very strong tendency to the deposit of fat. In East Africa we see thin, long-limbed stocks and fat stumpy stocks living in close proximity; but doubtless differences in feeding combine with differences of climate to produce these effects.[1]

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[1] Siemens, Ueber Linkshändigkeit, etc., "Virch. Arch.", 252, 1924.

[2] In Dr. Fischer's view right-handedness does not normally imply a like predominance of the right leg over the left. He holds, rather, that the left leg tends to predominate over the right—but that "left-leggedness" is a less conspicuous phenomenon than the customary right-handedness.—Translators' Note.

There are no fundamental racial differences affecting the distinction between the two sexes as to the way in which the subcutaneous fat is distributed to various parts of the body—but of course these peculiarities of distribution are hereditary, are under the control of the internal secretions.

An exception to the foregoing generalisation is furnished by the peculiar deposits of fat in the buttocks of Hottentot and Bushman women, giving them a marked protuberance in these regions, which is termed "steatopygia". This corresponds anatomically to the fatty hump in many species of bovines, and to the fatty buttocks and fatty tail in many races in sheep. Steatopygia makes its appearance regularly and typically, the fat being limited to the rump and to the upper parts of the backs of the thighs, the rest of the body being in some cases quite thin. It is a genuinely racial characteristic, restricted to the female sex, and hereditary. In half-castes resulting from the crossing of Hottentots with Europeans, well-marked steatopygia has not yet been observed, but only a strong tendency to a fatty deposit on the hips, thighs, and rump.

Among the peculiarities of bodily form, reference must be made to the differences in the female breasts. In elderly negresses, the breasts assume the form of a flaccid bag, this being to some extent a hereditary racial peculiarity, but to some extent the outcome of extremely prolonged lactation. (Negresses will continue to give the breast occasionally to children of five or six years of age, when the organ is properly speaking in function for the service of children born later.)

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[1] Schrötter, Tagebuchblätter des Herzogs zu Braunschweig, Vienna, 1915.

In the Bushman women, the breasts are placed exceptionally high, and much nearer the armpits than in women of other races. Among women of our own race, the position and to some extent the size of the breasts may be hereditary.

Certain other and fundamental differences between the general bodily structure of individuals would appear to be independent of nutritive influences, and to be the outcome (in part, at least) of hereditary determinants. Such characters are said to be "constitutional". Later in the present work (Section III) we shall consider the precise significance of this term "constitution", especially in relation to morbid phenomena and conditions. Here we are concerned only with differences between healthy individuals. There are very marked differences in the density or delicacy of the bones, the connective tissue, and the fat; in the strength and tension of the muscles; in the shape, curvature, and elasticity of the thorax; in the direction of the ribs; in the length or thickness of the neck; in the size of the head, the belly, the hand, and the foot; and in numerous other points, ranging down to inconspicuous trifles. One very obvious fact is that particular characters of the sort will sometimes combine to form special "types". There are, of course, transitional or mixed types, so that it is occasionally difficult to assign a particular individual to a particular type; but, speaking generally, definite combinations of characters are so common that we are fully entitled to speak of constitutional types.

In the description of such types, observers will differ one from another as to the amount of attention they pay to particular characters, with the result that somewhat different conceptions of the types are formed, and there are divergencies of nomenclature. French authorities distinguish between respiratory, digestive, muscular, and cerebral types. In the respiratory type the thorax is well arched, the limbs are delicately formed, the general growth is slender, and the head is small. In the digestive type we see a thick, barrel-shaped body, thick limbs, and a short neck. In the muscular type we have very muscular arms and legs. In the cerebral type we have, side by side with a comparatively feeble development of the trunk and limbs, a strongly arched cranium with a high forehead. These types already become plain in childhood.[1]

The usual German classification is somewhat different, though on the whole the types correspond pretty well.[2]

We distinguish:

1. the asthenic or leptosome type (see Fig. 28, facing p. 152);
2. the pyknic type (see Fig. 29, facing p. 153);
3. the athletic type (see Fig. 30, facing p. 153).

Persons of the asthenic or leptosome type are tall and slender; they have narrow, drooping shoulders, a long, flattened thorax, thin and poorly musculated limbs, and a skin that lacks blood. Persons of the pyknic type are comparatively stumpy, with a short, bull neck, a barrel-shaped thorax, a big belly, and a tendency to deposit fat in the rump, the neck, and the head, but less so on the limbs. Conversely, persons of the athletic type have broad and square shoulders, powerful extremities, large hands and feet. I have described these types only in outline, neglecting minor peculiarities which are often extremely characteristic. In women, the same types are distinguishable, with slight differences peculiar to the sex.

Sometimes the types shade into one another. When the asthenic type is pushed to an extreme, we get the morbid "phthinoid" type, but no sharp line can be drawn between the two. We might, for instance, make subdivisions within the asthenic type; and if we were considering gradations tending in the other direction, we might speak of oligosthenic and eusthenic types (rather more powerful, and considerably more powerful). Thus the eusthenic type would shade into the athletic.

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[1] Chaillon and Mac-Auliffe, Morphologie médicale, Paris, 1912—Mac-Auliffe, Développement-Croissance, Paris, 1923.

[2] Kretschmer, Körperbau und Charakter, fourth edition, Berlin, 1925.—English translation by W. J. H. Sprott, Physique and Character, London, 1925.

What is the significance of these types? I shall not enter into the question of their relationship to particular diseases, inasmuch as that will come up for consideration later (see below, p. 462). In popular tradition, thickset and short-necked persons are supposed to be prone to apoplexy, and slender persons with a narrow build to be prone to consumption. A basis of sound observation underlies these opinions (see below, pp. 308 et seq. and 391). Much more surprising, however, is the fact that the types are closely related with particular mental dispositions. In Germany, from two thirds to three fourths of schizophrenics are of either the asthenic or of the athletic type, whereas of persons suffering from the circular type of mental disorder (the manic-depressive group) from two thirds to three fourths are pyknics. We can go further and can show that even mentally normal persons who, in respect of temperament or character, show certain indications pointing in the schizoid direction, are predominantly asthenic; and that those whose temperament, character, and mental equipment incline definitely away from the schizoid, are predominantly of the pyknic type. Thus the physical type of constitution and the mental predisposition are plainly connected one with another, though in a way we do not yet understand. It is a matter of everyday experience that such associations and relationships are hereditary—that in many families we can trace a special tendency to particular kinds of temperament and particular types of character (which may or may not attain a morbid intensity, for the line between the morbid and the healthy is here particularly hard to draw), and in association with this we can trace the hereditary transmission of the before-mentioned bodily characters.

Inasmuch as these types exist side by side in the racially mixed population of our homeland, and inasmuch as their peculiarities are transmitted by inheritance (whether wholly or only in part is as yet unsettled)—since, therefore, they comport themselves just as do the previously described "racial characters"—it is natural that we should incline to think that they are themselves essentially racial characters. This conclusion has, in fact, been drawn; and, seeing that the "constitutional" characters in question closely resemble the predominant types in certain European races, the constitutional types have been identified with these various races. The tall, narrow, asthenic (leptosome) form is said to be a characteristic development of the "Nordic" race (see below, p. 186); the pyknic type is said to be characteristic of the "Alpine" race; and the athletic type is said to be characteristic of the "Dinaric" race.[1]

However fascinating the idea may seem at first sight, it will not endure the test of criticism. In Sweden, among Nordics of almost pure race suffering from mental disorder, there are almost as many pyknics as in Upper Bavaria,[2] and matters seem to be on the same footing in Italy and in Spain. On the other hand, the distribution of the insane and of the constitutional types within Germany, Switzerland, and Austria, does not harmonise in any way with the varying proportions in which the Nordic and the Alpine races respectively contribute to the population of these countries, though there should be such a harmony if the constitutional types were the expression of descent from particular races.[3] We must infer, therefore, that no such connexion exists. These types are found in all races. Within each race, consequently, there must be hereditary stocks formed by the carriers of particular constitutional bodily (and mental) characters, so that we may say that the racial type is dressed up in particular constitutional trappings. Hence there are asthenic Alpines, pyknic Alpines, and so on, each type clearly distinguishable from the other. But the Nordic pyknic will also be distinguishable from the Alpine. We may invert the foregoing proposition, and may say that the constitutional type contemplates us from beneath the particular racial trappings! We do not as yet know how, in these admixtures, race and constitution influence one another, and there lies open here a vast field for new investigations. Nor have any studies yet been made to show exhaustively how far the various constitutional types are represented in non-European races. Baelz, indeed, showed long ago that among Japanese there exist two main types, a fine and a coarse. Kretschmer has himself described pyknic and asthenic individuals as seen by him among Egyptian students.

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[1] Stern-Piper, Kretschmers psycho-physische Typen und die Rassenformen in Deutschland, "Arch. Psych.", vol. LXVII, 1923; also "Zeitschr. Neur. und Psych.", vol. LXXXIV, 1923, and vol. LXXXVI, 1923.—Pfuhl, Die Beziehungen zwischen Rasse- und Konstitutionslehre, "Zeitschr. für Kons.", IX, 1923.

[2] Henckel, Konstitutionstypen und europäische Rassen, "Klin. Wochenschr.", 1925, no. 45; Studien über den konstitutionellen Habitus der Schizophrenen und Manisch-Depressiven, "Zeitschr. Kons.", XI, 1925.

[3] Rohden, Ueber Beziehungen zwischen Konstitution und Rasse, "Zeitschr. Neur. und Psych.", vol. XCVIII, 1925.

In conclusion, and to throw a little more light upon these obscure conditions, I may mention that there are additional types of constitution to those mentioned above, but that these other types incline much more towards the morbid. Following Kretschmer, we can class them together as "dysplastic". They are the outcome, in part of a disturbance of the functioning of the endocrine glands (concerning which we do not know whether it is hereditary as well as congenital), and in part of factors which are still outside our ken. Among these we have as sub-classes "hypoplastic-infantile" types, that is to say types in which the adults display infantile under-developed bodily characters; eunuchoids, persons whose type has been altered by endocrine disturbances, so that they display polyglandular fat-abnormalities, and incline towards hermaphrodism, being endowed with some of the characters appropriate to the other sex; cretinistic forms; and so on. All these sub-classes show a bodily form typical to each, and certain elements in their make-up are doubtless hereditary; but the types have nothing to do with race in the ordinary sense of the term. We see, then, how greatly the investigation of heredity factors must be complicated by the circumstance that in each individual the racial types determined by racial heredity factors tend to be obscured by the interlacing of the no less definite characters belonging to the various constitutional types.[1]

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[1] The literature dealing with constitutional types is so extensive that I must be content here to mention the authors of certain well-known monographs: Bauer, Mathes, Martius, etc.

13. PHYSIOGNOMY

The characters summed up under the comprehensive term physiognomy vary to an extraordinary degree from race to race. Some of these characters are obviously hereditary. The nose varies in the size and shape of all its parts. The general shape of the nose is estimated in accordance with its length, that is to say the distance from the fronto-nasal suture to the labio-nasal angle, and its width, that is to say the distance between the alae nasi. Out of these elements we make a nasal index, and distinguish between narrow, medium, and broad noses. In noses of the narrow type, the nostrils are small slits running antero-posteriorly; in very broad noses, on the other hand, the slits of the nostrils are laterally directed. The bridge of the nose may be straight, concave, or convex. When convex, it may exhibit an equable curve; or it may have a sharp forward bend in the middle, being then termed aquiline; or, finally, it may run fairly straight from above downwards until near the lower end, when the point of the nose bends sharply towards the upper lip, thus producing what is known as the hawk-nose.[1] The study of racial crossings has shown that at least four heredity factors must be at work in the moulding of the nose. The shape of the bridge, that of the wings of the nose, that of the root of the nose, and that of the point of the nose, may be separately transmitted by inheritance. A prominent and convex nose is dominant over a broad and less projecting nose. This applies alike to crossings between Europeans and Hottentots and to crossings between Europeans and Jews.[2] On the whole, the shape of the nose is very little subject to paratypical influences, so that the observation of this feature is of exceptional importance in racial diagnosis. Particular shapes of nose and of face are often inherited together; there is also a remarkable tendency to the coupling of the heredity factors upon which the formation of the various separate characters of the nose depends.

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[1] These differences in the shape of the nose are anatomically dependent upon differences in the bones, the cartilages, and the soft parts. See Eickstedt, Beiträge zur Rassenmorphologie der Weichteilnase, "Zeitschr. Morph. Anthr.", vol. XXV, 1925.

[2] Salaman, op. cit.

When, as the outcome of crossing, the customary coupling of characters fails to appear, we get shapes of nose which at the first glance seem to us disharmonious. The same remark applies to the physiognomy as a whole. Inasmuch as the separate characters that combine to make up a physiognomy are capable of being separately inherited, so that they may be either transmitted as a whole from one parental side or can appear as a mingling derived from both sides, we have the possibility of the production of what strikes us as a racially harmonious type or of what seems to us an unbeautiful and disharmonious countenance. Often enough, when in ordinary life we encounter some one in whom a particular element of the face strikes us as uncongenial, observation of this person's parents or grandparents will show that into the racially harmonious face proper to one parental strain there has been introduced a trait peculiar to the other parental strain. For instance, a man whose face is long and narrow may have inherited a small snub nose from the maternal side; or a girl with a small, rounded face, may have it disfigured by a nose that is far too large which she has inherited from her father. That, again, is why an upper lip sometimes strikes us as much too short. Nature has to improvise a face out of the materials placed at her disposal by heredity, and these are often enough unsuitable for the making of a pretty mosaic! This probably accounts for many of the instances in which the jaws are too small for the teeth, so that when these are cut they force one another out of line. Here, once more, there is an extensive field for the study of mendelian heredity.

Before leaving this question of physiognomy, special reference may be made to the lips. A peculiar thickening of the lower lip with a marked prominence of the whole region of the chin (progenia) is a well-known phenomenon, which appears to be dominant in inheritance. It has been carefully studied as of frequent appearance in the male scions of the Habsburg line.[1] A marked tumidity of the lips, making the red part of the upper and the lower lip especially broad, is a hereditary character in Negroes. In African Pygmies, on the other hand,

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[1] Haecker, op. cit., and "Zeitschr. ind. Abst. und Ver.", 1911.—Strohmeyer, "Arch. Rass. Ges. Biol.", 1911–1912.

and in many of the Malay stocks, we find lips which are merely thick and not tumid as a racial character. The individual shape of the lips (narrowness, shortness of the upper lip, etc.) is manifestly hereditary in our own stock. Apart from gross changes artificially induced, for "decorative" purposes, in the wings of the nose and in the septum (perforation), the upper and lower lips, the lobules of the ears, etc., we know of no environmental influences which affect the separate parts of the face. We have already considered how far such influences modify the face as a whole (see above, page 127).

14. PHYSIOLOGY

It is self-evident just as the anatomical structures are hereditarily determined, so likewise are their reactions (that is to say, their functions). Thus we plainly see as a character common to members of a particular family, in one case a general robustness, in another marked nervous energy, in another great muscular strength, in another an excellent digestion, in another longevity—or the reverse of all these. This opens up the problem of the hereditary constitution (see below, pp. 306 et seq.). As regards details, I may refer to the easily observed characters of early or late puberty, the early or late onset of senility, and to longevity or the reverse.[1] Such physiological peculiarities as the age at onset and cessation of menstruation are likewise inherited.[2] The inheritance of the tendency to give birth to twins is discussed below on pp. 321 et seq. The likeness or the absence of certain chemical reactions in the blood can also be shown to be hereditary.[3] The "serological" method of investigation offers favourable prospects in this field. The presence or absence of various definite and easily distinguishable bodies and antibodies in the blood enables us to recognise differences between individual human beings. The frequency of some of these substances in the blood varies from race to race. Even when we confine our attention to Europe, there appear to be differences in this respect between members of the Nordic, the Alpine, and the Mediterranean races. These researches are only in the initial stage, but they are full of promise. The most striking contrasts are those between Europeans and Mongoloids; the Negroids are nearer to Mongoloids than to Europeans; and, on the other hand, strikingly enough, the Indians are nearer the Europeans. The data obtained so far are extraordinarily interesting, but great caution must still be used in drawing conclusions from them.

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[1] Friedenthal, Sonderstellung des Menschen, Berlin, 1925.—Pearl, On the Inheritance of Duration of life, "Papers Dep. Biom., Johns Hopkins", no. 89, 1925.

[2] Bolk (Untersuchungen über die Menarche bei der niederländischen Bevölkerung, "Zeitschr. Geb. und Ggm.") shows that in Holland blonds on the average begin to menstruate ten months earlier than brunettes; also that in recent generations the age of puberty has been reached a year and a half earlier than formerly. A. Schreiner found the same thing in Norwegian women. There is also unmistakable evidence that the age at the first onset of menstruation tends to be transmitted from mothers to daughters, without any paternal influence.

[3] Dungern and Hirschfeld, Ueber gruppenspezifische Strukturen des Blutes, "Zeitschr. Immun.-forsch. und exp. Therapie", vol. VIII, 1911.—

The pitch of the singing voice—bass, baritone, etc.—is likewise a mendelian character. Bass in the male and soprano in the female seem to correspond each to the other, and to be dominant in inheritance. The baritone and the mezzo-soprano voices appear to be heterozygotic, and the tenor and the alto voices to be recessively homozygotic. There is a good deal of evidence to show that among Nordics there are more basses and among Mediterraneans more tenors.[1]

15. CONCLUSION

The picture given above of the so-called normal variations of human beings is an extremely complicated one. Almost all the shapes and structures of the human body differ more or less from individual to individual and from group to group. What we want to know is, which variation or how much of such a variation is hereditarily determined; and which variation or how much of such a variation is produced by environmental factors, is, that is to say, peristatic. As a matter of fact,

Hirszfeld, "Klin. Wochenschr.", 1924, p. 1180.—Bernstein, ibid., p. 1495—Steffan, "Arch. Rass. Ges. Biol.", vol. XV, 1925, and "Zeitschr. ind. Abs.", vol. XXXVII, 1925.—Halber and Mydlarski, "Zeitschr. Immunitätsforsch.", vol. XLIII, 1925.—Steffan, Weitere Ergebnisse, etc., "Arch. Schiffs- und Trop.-Hyg.", vol. XXIX, 1925, and "Mitt. Anth. Ges.", Wien, vol. LVI, 1926.

[1] Bernstein, Zur Statistik der sekundarischen Geschlechtsmerkmale beim Menschen, "Götting. Nachr. math. phys. Kl.", 1923, and "Sitzber. preuss. Akad. d. W. phys. math. Kl.", 1925.

we never find the hereditarily determined element, the idiotypical element, manifesting itself in all its purity, for it is always modified by paratypical influences. The idiotypical characters, exclusively, are racial; they are the only ones which can be hereditarily distinguished from others and classified as peculiar to genealogically associated groups; and it is therefore necessary, by careful analysis, to differentiate the idiotypical constituents from the paratypical. What has been said above will show that for racial description and classification the most important characters are: the colour of the eyes and of the hair; the colour of the skin; the Mongolian spot; the growth of the hair; the shape of the nose; the folds of the eyelids; the shape of the lips and other physiognomical details; serological distinctions; stature; the shape of the cranium; the shape of the face. In the case of numerous other characters, we are still too uncertain about the differentiation of their racial hereditary from their acquired elements to use them for the purposes of racial diagnosis. This applies to the proportions of the body; to many details in the skull and the bones of the extremities; to variations in the muscles, the papillary ridges of the palm of the hand and the sole of the foot, the form of the teeth; etc., etc. But the characters classed in the former category suffice to show beyond the possibility of dispute that plainly inheritable differences exist among human beings which enable us to divide them into the groups termed races.[1] By crossing, these distinctions can apparently be obliterated, so that in wide geographical areas populations may come into being which seem to represent a continuous transition from one race to another. In reality, however, the races are sharply delimited. But within a race, or a racial mixture, there often arise peculiar "types", the result of cross-breeding and minglings of blood, whose origin and significance will be discussed below (p. 182). In the next chapter, however, we shall consider the general question of racial origins and racial biology, before proceeding to a more detailed description of the races of man ("anthropography").

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[1] In "Völker, Rassen, Sprachen", Berlin, 1922, Luschan, who writes for the general reader, denies this, and refers all the differences between races to the working of environmental factors. But he gives no proof whatever of his contention.

CHAPTER SIX

RACIAL ORIGINS AND RACIAL BIOLOGY

1. RACIAL ORIGINS

The origin of the human varieties, of the so-called "races", goes back to the origin of man in general. By his whole organisation man indisputably belongs to a special order of mammals, which includes all the monkey kind as well as himself—the order known as the primates. Within the order of primates, the anthropoid apes and the "hominids" (including both the extinct and the extant forms of man) constitute a separate group. Towards the close of the tertiary period there occurred an extensive evolution of the anthropoids and the hominids. The fossil remains of anthropoid apes show that in this tertiary period they were widely distributed, ranging from China and Hindustan across the whole of Europe and as far as South Africa. A main centre of their radiations, and perhaps of their origin, is North East Africa, where extensive discoveries in the Fayum (Upper Egypt) have given us valuable insight into the modifications of the monkey kind in general. The tertiary development of the anthropoid apes shows that at that time they existed in greater numbers than to-day and were more widely generalised. To some extent these tertiary apes died out, and to some extent they developed into the contemporary anthropoid apes. Of these last, the orang, the gorilla, the chimpanzee, and the gibbon, has each developed in a different direction and has become adapted to peculiar circumstances of life. To this general development of the primate stock belongs also the line from which extant man is descended. It follows, therefore, that man must not be regarded as a descendant of any of the contemporary anthropoid apes, but that his stock like that of these anthropoids is a descendant of a more generalised and primary form, whose organisation has in certain respects been better preserved by some of the extant lower monkeys than by the anthropoids. Naturally, however, the lower monkeys have also undergone specialised developments of their own. We see, then, that we must expect to find (and do actually find) fossil forms resembling extant man more closely than do the contemporary anthropoids. Such forms, though not perhaps direct ancestors of the men of to-day, must be close to the direct line of human descent, from which the monkeys have branched off at an earlier stage. That is why we class these close collaterals of our ancestors with our true ancestors and with extant man, separately from the other primates, as hominids. The most important of the fossil hominids is Pithecanthropus erectus, whose remains were discovered in Java. The geological deposits (later diluvial) in which Pithecanthropus was found indicate that he is not likely to have been a direct ancestor of ours. Moreover, extremely careful anatomical study of the skull and the femur has shown that he cannot be regarded either as a human fossil, or as an anthropoid in the contemporary sense of the term. We have, therefore, in Pithecanthropus, not indeed an intermediate form between extant man and the monkeys. We have what theory demands, a more primary, more generalised form endowed with the characters neither of contemporary monkeys nor of contemporary man, but with characters from which both the human and the monkey characters can be derived. Pithecanthropus is a form which has remained arrested on the way to become man, or perhaps it would be better to say upon a closely adjacent side path![1]

The factors which produced man out of the ape-man must have been something of this kind. The starting-point was a "primatoid" creature, one with feet adapted for climbing trees, semi-upright in his posture and fitted for life in the tropical primeval forest. While life in this forest continued, our arboreal creature would never have become modified into one adopting a wholly upright posture, for such a change could only be induced by external compulsion—that is to say by the disappearance of the primeval forest. By secular changes in the environment, the creature must have been forced to abandon arboreal life and to become adapted to life in the open plains. The most important change in his organisation was unquestionably the origination of the foot. It has been said, and rightly, that man became man with the growth of the foot. Adaptation to a life in open parkland or in savannas compelled this ape-man to adopt a wholly upright posture. Twice in the monkey stocks evolution has witnessed such a transition from forest life to life in regions with few trees or none. Once this happened in the line which leads to our human race, and once it happened in the line leading to the baboon. In the latter case, the creature abandoned the semi-upright position to assume that of a quadruped once more. The baboon is typically adapted to life among rocks, and hardly ever climbs trees. This is plainly indicated by its structure, which differs so much from that of the other monkey kinds. But, conversely, the other branch, that of our own ancestors, became wholly adapted to a bipedal life. We cannot here discuss the mechanical modifications which took place in the ape-foot during its transformation into the arched foot of the human being.[1] Once the first impetus had been given, once the erection of the body upon the hinder extremities had taken place, the subsequent changes in that body were necessary consequences. For statical reasons, the human spine had to develop its characteristic curves and a sharp bend at its junction with the pelvis (the sacral promontary), and the pelvis had on either side in its upper part to assume its present wide "shovelled" form. Now the skull could become heavier, being balanced upon its supports beneath, and no longer hanging obliquely at the front end of the vertebral column as it does in quadrupeds. The exciting cause of all this was an extremely strenuous struggle for existence, with consequent rigorous selection. As long as the creature was still living in the primeval forest, adapted to life there, and satisfactorily nourished by the abundant fruits of the forest, there could not occur the very rigorous selection of the kind we are now considering. But with the changing circumstances, such a selection was necessarily initiated. New qualities had to be bred; the creature's form had to adapt itself to the new surroundings. On the one hand, the adoption of life in the open plains exposed these ancestors of ours to a number of new enemies, predatory creatures of the plains, already adapted to life there; on the other hand, the gaining of nutriment under the new conditions must have been extremely arduous for the creature now undergoing modification. The sparse fruits of the open steppe would no longer suffice him. He had more and more to adopt a flesh diet, and must have been hard put to it to keep himself alive. Every slight improvement in the stability of his gait, now that he had to stand firmly not only in repose but also as a basis for the act of striking and throwing, had become of vital importance to him. Now, likewise, selection was actively at work upon his intelligence. In the whole monkey group, as compared with the other orders of mammals, the intellectual capacities, grounded upon an exceptional size of the brain, are already considerable. Thus there existed a suitable start for the breeding of a very large brain, and in Pithecanthropus we find that the cranial capacity has already increased to 1000 cubic centimetres, whereas in the extant anthropoid apes it never exceeds 600 cubic centimetres. But such an extensive and continuous increase in the weight of the brain was only rendered possible by the adoption of the upright posture.

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[1] Cf. Schwalbe's brilliant account of the matter in the volume Anthropologie of "Kultur der Gegenwart", 1922. Recently some authorities have disputed the theory that the skull and the femur found in Java belonged to the same creature. If their objections are sustained, what has been said above will still apply, with certain reservations, to the skull.

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[1] Weidenreich, Der Menschenfuss, "Zeitschr. Morph. Anthr.", 22, 1921.

Now came the first acquirements which we regard as the peculiar privileges of man. Presumably the rudiments of speech date from this period; but, in addition, it was already an immense step forward when the developing human creature began, not merely like the extant monkeys to appreciate the comfort of a fire, but by "feeding" it to make sure that a fire once obtained should be kept permanently alive. Out of this developed the art of "fire guarding", and therewith man assumed a position on a higher level than that occupied by any other members of the animal kingdom. Now the warming of his habitat and the preparation of his food could become purposive activities. Notable advantages were given him in the struggle for existence by the ability (especially in the tropics!) to make use of the power of heat to promote the keeping qualities of food, and of its power to make certain vegetable products (otherwise poisonous or indigestible) available for use as food. Thenceforward man came in great measure to modify his food deliberately, and doubtless at the same time to modify deliberately in connexion therewith his whole life, his reproductive activities, the organisation of a communal existence; thus gradually developing custom and law. More and more, from this time onwards, man must be biologically regarded as a domesticated creature.[1]

The whole process was so exceedingly complicated that we find it impossible to conceive its having taken place several times in like manner and thus to have led to the formation of similar final products. It can only have happened once for all. We have, indeed, good reason to assume that various kinds of primal anthropoid apes must have been exposed to the influence of marked changes in their environment, but that they were unable to adapt themselves to these changes rapidly enough, and therefore died out. The extant fossil remains of anthropoids show how much these creatures must have been thinned out by environmental changes. Only five of the species that have sprung from the common anthropoid-hominid root were able to survive all those geological and climatic changes—man and the four existing types of anthropoid apes, each of them peculiarly well adapted to its environment, but man most notably modified of them all because exposed to the most extensive changes in environment. We must, therefore, suppose that the evolution of man was a unified occurrence, taking place once for all and in only one place. Of course, we must not conceive this "place" as having unduly narrow limits. We shall probably not err in thinking of it as comprising a wide area, perhaps one as large as half Europe. Such a great region, throughout which the particular ancestral form of primates was equably distributed, must have been the subject of secular changes for tens of thousands of years, so that these primates were uniformly modified in the way above described. As to the geological date, the modifications must have occurred towards the close of the tertiary period. Most theoretical considerations in the realms of geology and zoology combine to indicate this, and the fossil record confirms the hypothesis.

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[1] See the references to Fischer's writings in the bibliography.

The effective factors of this whole evolutionary process were, as already said, the change in the environment, and the resulting change in the trend of selection with an intensification in its severity. The slightest modification which would be advantageous to the preservation of the individual, which would help him in obtaining food, in resisting his enemies, in perpetuating his kind, in maintaining his health, etc., would favour him (or her) as against competitors. It was this extremely rigorous selection which changed the generalised primate form to primitive man and then to man. New characters arose, and old ones disappeared. Thus it is that man has in his blood, as an expression of heredity factors, first of all, a number of proteids which are common to him and all the monkey kind, others which exist only in him and the anthropoids, and yet others which are peculiar to himself. These last are the outcome of this transformation. The vestiges of man in the Ice Age remain as tangible evidence of the gradual change.

They exhibit forms which no longer exist to-day, so that systematists often describe them as distinct species. Some of them are probably the direct ancestors of extant man. We can plainly recognise successive evolutionary gradations. The oldest geologically and the most primitive anatomically is the man of Mauer near Heidelberg (Homo Heidelbergensis). In sequence, both temporarily and anatomically, we have the men of the Neandertal type (Homo primigenius, as found at Neandertal, Spy, Krapina, La Chapelle, Le Moustier, etc.). These forms are markedly and clearly distinguishable from all contemporary men; every skull is indubitably proved by direct measurement to belong to this group. Then, in further succession, we get forms described as "transitional", but still belonging to the Ice Age: the Aurignac race; the so-called transitional forms of Brünn and Brüx, etc. (whose racial relations have not yet been fully worked out); the vestiges found at Oberkassel; those found at Predmost; and then those belonging to the Cro-Magnon race.

The Cro-Magnons border on the races of neolithic man, who, geologically speaking, belongs to the present day. All these transformations were likewise brought about by rigorous selection, in conjunction, doubtless, with the changes in environmental conditions which are summed up under the name of the Ice Age. Selection has continually maintained and modified the human form. Its effect upon the activity of the intelligence is shown in the development of civilisation, as manifested by thousands of stone, horn, and bone implements dating from the earlier Stone Age. They run parallel with the bodily evolution.

Thus we find that as man developed, almost simultaneously, or at any rate at an early stage of his development, a separation into varieties occurred. We may say, indeed, that the formation of species is at the same time the formation of races. Obviously, in the fairly large geographical area where the transformation of the pre-man into man took place, it would be impossible for the external conditions to have everywhere the same effect; and, conversely, the reaction of individuals and groups upon the external factors must have varied from place to place and from time to time. The whole group manifests variability, and in particular places particular variations must certainly have come into being. Thus from the very outset there must come into existence slight regional differences in man.[1] We may, indeed, assume that the most marked among the distinctions between the contemporary races of man must have begun very early in the history of the human race. It is probable that the three main stocks of Europoids, Negroids, and Mongoloids go as far back as this. Nevertheless it follows from the nature of the case that there could not have been a simple partition of man into two or three main stocks; but that, from the very first, side by side with these main stocks, subsidiary stocks must have come into existence, like the main stocks, but differing from them less than these main stocks differed one from another. That hypothesis accounts for the fact that, side by side, with the Negroes, there exist certain

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[1] From the foregoing account of the "becoming of man", it follows that these "slight differences", that is to say the new acquirements developing as racial characters, concern only secondary points. All the races of man are fully alike in respect of the characters which belong to man as contrasted with all other animals. Consequently the original primate form which became man did so simultaneously throughout the entire geographical distribution of that type. The origin of man, I repeat, took place once for all, out of a single species of primates. For instance, the delimitation of the hair of the head, the arrangement of the papillary ridges on the palms of the hands and the soles of the feet, the typically human composition of the blood serum, and many other characters, became those of the human kind simultaneously, everywhere, and once for all; they are alike in all human beings, and existed before racial differentiation occurred. But then, after that, there were changes in the hair, in the frequency of particular patterns in the papillary ridges, the appearance of particular serum elements in particular races, and so on.

This view accords with Schwalbe's, for that authority definitely expressed himself in favour of such a monophyletic and monogenetic origin of man. Klaatsch, on the other hand, inclines to refer the separation of the races of man to an earlier day and an earlier stage of evolution, when the forms concerned had not yet become men but were creatures ancestral both to the extant anthropoid apes and to the extant races of man. Such a view, which is termed polyphyletic, is most improbable. Lubosch makes a serious mistake in minimising the sharp distinction between the two views!

Melanesian groups, closely resembling the Negroes in many respects, but differing from them in a number of details. Hence the attempt at a perfectly systematic classification of the races of man is futile. As soon as mankind had reached the level above sketched, as soon as human beings had achieved a primitive culture, as soon, above all, as they had mastered the use of fire (there must have been a long, long period of fire-guarding before the period of fire-making began); as soon as, in other words, man was sufficiently developed to be regarded biologically as a domesticated creature[1]—phenomena arose resembling the ones which we can to-day observe in domesticated animals. New formations and racial distinctions arise through the appearance of idiovariations, and it would seem that in a state of domestication idiovariations occur with especial frequency, and, above all, that they are preserved more frequently by selective breeding. What we find in human beings as racial differences, exist (mutatis mutandis) in domesticated animals; and, conversely, nearly all the domesticated characters of our domesticated animals are to be found (mutatis mutandis) in man likewise. Especially let me remind the reader of the variations of the hair in respect of form, length, and colour; of the variations in the tint of the skin and the colour of the eyes, in the stature, and in the shape of the nose. As an example, I may refer here to the origination of the so-called white skin and of fairness. It is a matter of common observation that comparatively fair individuals appear from time to time in all races. Under natural conditions, however, a fair skin is dangerous to dwellers in the tropics, so that in those regions persons exhibiting such a variation are eliminated by natural selection. The tropical sun is so harmful to the human body when unprotected by clothing or by an ample supply of pigment that a fair and blond race can only arise in temperate regions. The fact that the blond inhabitants of northern Europe exhibit a general distribution of pigment, above all in the iris, exactly like that of the fair domesticated animals, but quite unlike that of animals belonging to the polar zone, is plain proof that we have to do here with a form resulting from domestication which is able to maintain itself in temperate climes, whereas similar idiovariations have always been eliminated in the tropics by natural selection and are still so eliminated there whenever they arise. Natural selection continues to work upon contemporary man, above all upon those stocks which stand at a low level of civilisation. Human beings still living in a state of primitive culture remain strongly exposed to the influence of natural selection; especially as regards the acquisition of food; as regards their capacity for undertaking great marches, for enduring heat, thirst, and hunger, for withstanding the effect of considerable losses of blood, for getting along passably with food difficult to digest, for easy childbearing and abundant power to suckle their children; in the matter of having vigorous limbs, acute and normal senses, and adequate intellectual powers adapted to the circumstances of their lives. From time to time the rigour of natural selection is, for such human beings, intensified to the uttermost by the inevitable recurrence of periods of drought, and of famine due to the dying-off of game. Nature, therefore, perpetually sees to it that the race shall remain at the acme of efficiency. Only with the advance from savagery towards civilisation, and still more when civilisation reaches a high level, is the rigour of natural selection diminished (as will be shown in the third section of this book), to some extent deliberately, but in the main unwittingly. Superadded to the effects of natural selection, which obviously accounts for many of the differential characters in the extant races of man, have been the effects of sexual selection. This latter presumably accounts for such characters as the racial growth of the beard, for steatopygia in Hottentot women, and for various other peculiarities.

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[1] Critics have objected that this concept of domestication does not apply to man, seeing that the essential characteristic of domesticated animals is that in respect of their breeding they are subjected to the will of man. But what I mean to imply by the use of the term is no more than this, that in biological behaviour, in changes of food, reproduction, the choice of climate, etc., domesticated man comes to resemble the domesticated animals, with the same result, that marked variability ensues. That is why I have spoken of man as, biologically considered, being subjected to the influences of domestication.

2. RACIAL BIOLOGY

A knowledge of the foregoing factors, as they have been at work in the production of races, throws much light upon the domain which we may speak of comprehensively as racial biology. The various races of man have intercrossed one with another in manifold ways and throughout numerous regions. The problem of racial crossing has first become soluble since we have gained a knowledge of the laws of mendelian separation. It can be demonstrated that all the effects of racial crossing are subject to these laws. I shall not attempt to trace once more here the crossing of separate characters, and shall be content to refer to a few phenomena of a more general kind. First of all it is proved that all the races of man can interbreed, and that the hybrid offspring remain fertile. There seems to be scarcely any diminution of fertility in human hybrids, for the cases of reduced fertility in mulattoes can be otherwise explained. Especially important would appear to be the demonstration afforded by an exact study of racial crossings in the light of mendelian laws, that there is no "prepotency" as a heritage of particular races, though in former days such prepotency was generally assumed to exist. The assumption that when certain so-called savage races (here people think also of the Negroes) are crossed with the co-called civilised races, the savage type will always tend to "break through" in the mixed breeds, is certainly erroneous. There is no such thing as a general predominance of the hereditary characters of one race or another, for in human racial crossings, just as in thousands of experiments upon plants and animals, this or that particular racial equipment proves dominant or recessive in inheritance. If a race happens to possess many such dominant characters, then, in the hybrid population, its characters will be more conspicuous than will be the characters of the other stock, in which recessive traits are more numerous. That explains why, when Negroes and Europeans are crossed, the resulting offspring of mixed blood are most often dark-skinned and dark-haired. The erroneous view that the characters of particular races tend to "break through" in the mixed breeds is fortified by a psychological blunder—especially where the crossing of the European races with the Jews is concerned. In the grandchildren (F_2) and in following generations people often note that particular "Jewish" characters are conspicuous although perhaps the members of F_1 did not appear Jewish at all. The tendency is, however, to overlook all these instances in which, conversely, only non-Jewish characters are obvious, and in which, therefore, to be logical, people ought to talk of a breaking-through of the non-Jewish elements. Precise investigations of crossings with Jews have shown that here, likewise, as must obviously be the case, what "break through" are merely particular dominant characters, such as the black hair, the convex nose, and perhaps one or two other traits in the physiognomy. On the other hand, in crossings between broad-nosed Jews of the eastern stocks (Ashkenazim) and North Europeans with a sharply-cut narrow nose, this Nordic nose is manifestly dominant.[1]

New races can never be originated by crossing alone. All that crossing can effect is to create new combinations, for it is incompetent by itself to eliminate old characters. The disappearance of the old characters and the appearance of genuinely new ones can only be the outcome of selection. By selection, however, the new combinations may be dealt with in such a way that all the carriers of particular qualities disappear, and those only who exhibit new combinations remain. Then a new race will be the outcome of the crossing, but the actual causes of its production will be selection and elimination.

The question now, however, arises whether the process of hybridisation can itself originate genuinely new characters in addition to new combinations: and if so, whether these new characters are the expression of a change in the hereditary equipment (idiokinesis) dependent upon the hybridisation (a phenomenon whose occurrence we have not yet been able to prove with certainty, or to explain if it occurs); or whether they are nothing more than paravariations, due to the hybrid character of the metabolism, or to some similar change. On page 152, I referred to the fact that in racial crossings the stature of the mixed breeds may, on the average, considerably exceed that of either parental race. I must also point out that in Malay-Tamil crosses, and also in Chinese-Malay mixed breeds, in Anglo-Indian Eurasians, and in European-Hottentot crosses, there has often been noted a considerable and at times very great elongation of the face, so that the cross-breeds have much longer and narrower faces than those of the members of either parental race. In Europe, too, when northern Europeans are crossed with Jews, with southern Europeans, with Gypsies, and with Lapps, this marked elongation of the face is apt to occur. We see it very frequently in the princely families of Europe, which are of extremely mixed blood.[1] At the same time the cranium seems to become relatively wider, but it has not yet been ascertained whether this is due to an absolute increase in breadth (as would appear likely) or to a reduction in the length.[2] This is a very interesting phenomenon, and it deserves careful study in families with an admixture of foreign blood. Kindred thoughts are aroused by the circumstance that the South Germans, who are of extremely mixed Nordic and Alpine stock, are notable for their long faces and broad crania as compared with the other Germans. When parts of individuals or the individuals as a whole become larger or stronger in consequence of hybridisation, we speak of the phenomenon as "luxurisation".

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[1] Lenz ("Arch. Rass. und Ges. Biol.", 16, 1925) refers to the possibility (which is unsupported by actual examples) that in racial crossings certain hereditary traits may favour the likelihood that a particular spermatozoon will fertilise the ovum. If this were to happen, selective phenomena would of course arise, and the hereditary characters of the two races would be unequally transmitted.

Injury to the constitution may also result from hybridisation. Mulattoes would appear to show less resistance to all kinds of diseases and noxious influences than the members of either parental race. Cross-breeds between Scandinavians and Lapps also seem more liable to tuberculosis than the parent stocks.[3]

This is a matter of enormous importance to medical science, but as yet very little light has been thrown on it. We do not know, for instance, whether constitutional infirmities or other morbid phenomena are produced by what we may call incompatibility of certain heredity factors, when races more closely akin than the before-mentioned are crossed. It seems possible, at any rate, that the crossing of European races may sometimes have this effect.

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[1] Hagen, Boas, Lundborg, op. cit.

[2] Hauschild, Die kleinasiatischen Völker und ihre Beziehungen zu den Juden, "Zeitschr. Ethnol.", 1920–1921.—Fischer, Rehobother Bastards (loc. cit.).—Leys and Joyce, "Journ. Anthr. Inst.", vol. XLIII, 1913.

[3] Lundborg, op. cit., and "Hereditas", II, 1921.

Perhaps instead of "luxurisation" being the result of such a crossing, "pauperisation" may ensue!

Disharmony in the mental domain might (insofar as it is not due to environmental causes, such as education or social conditions) be brought about by a failure of the hereditary mental equipment of the two races to adapt themselves properly each to the other.[1] We must, however, always bear in mind the possible working of such social influences as have been shown to be probably operative in the case, for instance, of the mixed population of Thuringia.[2] It has also been shown that among the insane in Swedish asylums a dissociation of the normally associated colour of the eyes and of the hair is not commoner than in healthy Swedes (recruits), and this affords a very important indication, though of course it does not amount to strict proof.[3]

We see, then, that numerous phenomena worthy of note arise exclusively as a result of the process of hybridisation.

The most thoroughgoing racial admixture occurs when two whole nations are mingled, and here there arise the most intimate mutual relationships between nation and race. The concepts nation and race have not necessarily anything in common. By a nation or people we understand a group of human beings united by the elements of a common civilisation, the most important of these elements being the common speech; but when we speak of a race, we mean human beings with a like hereditary equipment. Nevertheless the concept of race is of the utmost importance in connexion with the concept of nation. The sustainers of a particular national culture are, of course, individuals belonging to a particular race or to a particular mixture of races. The results of a mingling of two nations—which generally arises through the conquest of one nation by another, the former then imposing its nationality upon the latter; but which sometimes ensues through gradual and peaceful permeation—are of peculiar and fundamental importance to the racial composition of all extant nations. The most conspicuous occurrence of the kind is that in which the people of one nation spread themselves over another nation as a conquering stratum. In such a case, the former nation may be racially akin to the latter, or may be of an entirely different make-up. Again, the language and the national tradition of the two may be very much alike, or may be extremely different. The outcome of the process will be likely to differ a great deal in the respective cases. Where there is much divergence, the cultural elements of the two will come into conflict, so that, to speak metaphorically, there will be a struggle for existence between them. The language of the conqueror nation may more or less completely replace the language of the conquered, but the conqueror nation will almost certainly adopt some of the linguistic elements of the conquered, thus introducing modifications. The result may therefore be that in the area concerned an entirely different language will come to be spoken. We find that in extensive regions throughout contemporary Europe, Indo-Germanic tongues are spoken where it can be shown that in earlier days non-Aryan languages prevailed. Similar results occur in respect of the other acquirements of civilisation, both mental and material. But for a long time the importers of the new language may refrain from mixing their blood with that of the subject people, and may continue to reproduce themselves as a pure line. The line will be kept most conspicuously pure when conquerors and conquered are held apart by the racial pride of the former, and by barriers of custom, morality, and law. Such influences were particularly trenchant in ancient Hindustan, where the various strata of the population were severed by the supposedly insuperable barriers of caste. It is noteworthy that "varna", the Hindu for caste, denotes colour, for the conquerors were proud of their fair skins as contrasted with the darker skins of the conquered. But even when the barriers of caste were reinforced by the prescriptions of religion, and where law and custom strictly forbade a mingling of the two stocks, they were never really segregated from one another. Sooner or later, slowly or quickly, drop by drop or in great streams, the two bloods were mingled. Then peculiar processes of selection got to work. If the conquering invaders were less perfectly adapted than the indigens to the climatic and other conditions of their new habitat (having themselves become adapted previously to very different conditions), natural selection would vigorously eliminate the individuals that inherited the unsuitable characters. Selective processes would further be promoted by social factors. Among almost all civilised races, and without exception in the western world, the upper classes are less prolific than the lower. Furthermore, a good many efficient males are eliminated by war, political persecution, and other influences. When these things go on for generation after generation, the inevitable result is that the sustainers of these particular hereditary characters will be weeded-out of the mixed population. Long before anything was known about such selective processes, and long before the laws of heredity had been elucidated, it had been noticed that, when two peoples were intermingled in this way, the original type peculiar to the locality tended to re-emerge in almost complete purity. Some observers have spoken of a resifting of the races. What happens, however, is not any peculiar process of inheritance, is not an incomprehensible "breaking through" of the old indigenous type, but a simple destruction of the ill-adapted immigrant elements by means of natural and social selection. The final upshot is the production of a new people with a new language, and with a civilisation new in other respects, though, speaking generally, the inhabitants of the region will be of the same bodily type as that of the indigenous people before the conquest occurred. Only certain traits, appearing in isolation here and there from time to time as the expression of isolated heredity factors making themselves manifest in accordance with mendelian laws—some of them unmistakably physical and racial—will disclose that in former days foreign elements were introduced into the population. Culturally, on the other hand, the foreign admixture will have been decisive, and only casual remnants of the old civilisation will survive.

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[1] Mjöen, Harmonische und unharmonische Kreuzungen, "Zeitschr. Ethnol.", LII, 1920–1921.

[2] Binswanger, "Archiv. für Psychiatrie", LXXIV, 1925.

[3] Henckel, Konstitutionstypen und Europäische Rassen, "Klin. Wochenschrift", 1925.

Such is not, indeed, the invariable sequence. The reverse may happen. If the conquering invaders are well adapted to the new biological environment, if the circumstances in which they now find themselves are suited to them racially as well as in other ways, they may, for practical purposes, replace the subject stock; even then they may take over many mental acquirements and cultural traits from the subjugated people, though these are foredoomed to elimination.

But the racial composition of a people is, in all circumstances, decisive (in conjunction with other conditions) as to its cultural and mental functional efficiency. Of course we must not overlook the fact that the rise or the fall of a nation in history is dependent upon a number of external factors, such as its geographical situation, its relation to its neighbours, their character and doings, and a thousand other circumstances, which historians are in general inclined to regard as the only ones of decisive importance. Unquestionably, however, in addition to these, racial endowments play a mighty part. The various races of man differ from one another to an extraordinary degree in mental respects no less than in bodily. Some races are highly gifted as regards imagination, vigour, intelligence, etc., these qualities being very variously combined, hereditary, and inalienable. Just as the environment modifies physical qualities paratypically, there can be no doubt that, as in the individual, so in the nation consisting of thousands upon thousands of individuals with their special racial endowments, the circumstances of life must modify in many ways the development of the hereditary mental equipment. But the endowments are themselves provided by heredity once for all; and, inasmuch as the nations are racially different, their mental gifts must also be different. The North American Indians were living for generation after generation in close proximity to deposits of copper, but they lacked the intelligence which would have made them technically competent to discover how to smelt copper or to alloy it with tin to make bronze. Many of the peoples of the South Sea Islands must be of the same antiquity, but whereas some of them have not advanced beyond the stage of the most primitive stone culture and remain hunters and gatherers, others have discovered for themselves how to till the soil with the use of the hoe. Again, the people of Asia Minor and Europe who advanced to the use of the plough and to the associated type of civilisation are no older than their neighbours the Africans, being merely different in their mental endowments. We are coming to recognise more and more clearly that racial factors, and especially hereditary mental endowments, although they work (it cannot be too often repeated) in conjunction with other factors, are among the most influential in determining the course of a nation's history. Now, it is precisely here that the importance of racial minglings becomes most conspicuous. In many cases, the conquering stratum will take over the leadership. It is no chance matter that so often after a vigorous mixing of two races and when through the invasion of a conquering stratum a new nation has been builded out of an old one (as happened in the formation of the Indo-Germanic peoples, of the ancient Greeks, of the Latins, etc.), a remarkable mental impetus, an unfolding of art and science, and a striking political advance, have been observed. Such a process is determined by the laws of heredity. New mixovariations have arisen in the mental domain. It is, of course, essential to such an outcome that the conquerors and the conquered should not diverge too widely each from the other in their mental endowments; that they should, as the phrase runs, "suit one another". No one can foresee whether a crossing will prove favourable or unfavourable in this respect. We shall only be able to judge by the results. It is essential to the economic, artistic, and political advance of a nation that it shall have a sufficiency of the endowments requisite for the sustainers of the functions concerned; and also that it shall contain a few persons of genius fitted to be leaders in the economic, artistic, and political domains. Obviously, moreover, the gifts of these leaders must take the form of intensifications or at least partial intensifications of qualities which are widely diffused throughout the nation—for otherwise leaders and led will fail to understand one another, as we can often see for ourselves in such cases to-day. When the mentally gifted leaders are lacking, a nation may still be able to endure the lack without obvious detriment so long as no dangers threaten from without; but as soon as the days of peril arrive, it will inevitably succumb. Thus we see that these anthropological questions play a decisive part in the destinies of a nation. For a nation may grow old although a race can never do so. The races of man no more grow old and die than do the races of lower animals or of plants. They can only be eliminated by selection, thus dying an unnatural death. What historians regard as degeneration, sickness, and ageing of a nation, what they look upon as the decline of a nation, are the outcome of a reversed selection of the racial constituents of the people concerned. When the hereditary carriers of the requisite gifts, the racial leaders, have been eliminated through the working of some process of selection, the people is no longer the same as it was in former days. If, whether through war or by peaceful methods, the racial composition of a nation has been modified by degrees in consequence of the immigration of new racial elements, and even if there has been no more than a change in the racial composition of the leading stratum, the mental functional capacity of that people will be different from what it was. Now though, as already said, historians will incline to look upon this new way of functioning (especially if it takes the form of an economic and political decline) as a manifestation of senility, it is in reality due to a racial change. The Greeks of the Age of Pericles were of different composition from the Greeks who trembled before the Roman emperors; and these, again, were different in composition from the inhabitants of contemporary Greece. Like considerations apply to the destiny and the nature of our own people in the course of the centuries, and the present fate of the German nation has just such an anthropological cause.[1]

These concepts, race and nation, come into mutual contact in yet another connexion. In every human group or nation or stock of persons united by a common national culture and above all by a common language, and still more within the sub-groups that are formed by religious, occupational, and other influences (notably by the localised influences of the natural features of the country), there prevails a certain amount of inbreeding, the groups and the sub-groups being to some extent shut off from one another by barriers. Within these groups and sub-groups, therefore, hereditary lines will be bred, and specialised types will arise. Such a process must have been going on from the first in all nations since they began to exist as such, differing in its intensity from place to place and from time to time. Within such an "island", such a "canton", such a "conventicle", the non-hereditary environmental influences must also vary from one to another, and this will explain the differences which the shrewd observer cannot fail to note in the anthropological characteristics of this and that section of a people who racially form a unified nation. Thus we distinguish Frisian, Lower Saxon, North Swiss, Swabian, and other "types"; and however much dispute there may be about details, there can be no doubt as to the main fact.[1]

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[1] In Racial Hygiene, the sequel to the present work, Lenz will refer to the literature of this subject, and especially to Gobineau, Grant, Seeck, etc.

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[1] A detailed study of the racial characters of all the European nations, beginning with our own German stock, is an urgent desideratum.—In this connexion the reader may consult a collective work now in course of issue, the first volume, published at Munich in 1925, being by Walter Scheidt, and entitled Allgemeine Rassenkunde.—A periodical for the study of these questions has recently been founded, entitled "Volk und Rasse", and is published at Munich.—Eickstedt speaks of the localised types of human beings that thus arise as "cantonal types". See Eickstedt, Gedanken über Entwicklung und Gliederung der Menschheit, "Mitt. Anthr. Ges.", Wien, vol. LV, 1925.

CHAPTER SEVEN

DESCRIPTION OF THE RACES OF MAN

(ANTHROPOGRAPHY)

THE systematic description of the various races of man is termed anthropography. It deals with the different groups in accordance with their anthropological characters, compares them one with another, and endeavours to arrange them systematically. The conventional divisions are out of date and comparatively worthless.

Of course every classification based, not only upon physical characters, but upon such matters as language, is radically unsound. As has already been shown, language and nationality can change without any corresponding change in the race. There is no necessary kinship between a Portuguese and a Swede because both of them speak tongues belonging to the Indo-Germanic family. The Negroes of the United States have lost all memory of their Bantu language and have adopted English as their mother tongue, but racially they remain Negroes none the less. In racial nomenclature, therefore, it is desirable to avoid the use of names which have a strongly national or linguistic connotation. The most logical practice would be to use names based upon purely physical characters, as is customary in zoology and botany. Still, there is no valid argument against the use of names based upon geographical distribution, which are perhaps more suitable for human beings than strictly biological terms, so long as they do not carry with them an unproved hypothesis as to origins. It would be superfluous here to give details of earlier attempts at classification.

The following anthropographical description runs, in the main, along geographical lines, though certain large anthropographical domains must be separately treated in view of particular racial connexions. Considerations of space have made it impossible to give more than a brief sketch of the extra-European races.[1]

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[1] For fuller details, consult the volume Anthropologie, in Kultur der Gegenwart, Leipzig, 1922. See also Günther's elaborate work, Rassenkunde des deutschen Volkes, eleventh edition, Munich, 1926; and Rassenkunde Europas, second edition, Munich, 1926. I regard as erroneous Günther's nomenclature of the races, and his classification into further main races than those here described.

1. EUROPE

Despite the numerous and extensive national migrations, both prehistoric and historic, which have taken place all over Europe, it can be shown that vestiges of particular races have maintained themselves fairly pure in certain areas. When the distribution of the most important anthropological characters is set out upon a map of Europe, the combination of these characters enables us to recognise clearly that even to-day the geographical distribution of particular races can be ascertained (see Fig. 31 on the next page). It is self-evident that the extant geographical frontiers of Europe are not racial frontiers, for the southern European races spread over from south-eastern and south-western Europe into Asia Minor and Africa respectively; and in like manner, as we shall have soon to discuss in fuller detail, there is a spread-over of races from Central Europe into Asia, and conversely of Asiatic races into Europe.

The north and north-west of Europe and far into Central Europe, with the coastal region of the North Sea and the Baltic in especial, are mainly occupied by what is known as the Nordic race. That race is in almost exclusive possession of certain parts of Sweden, and also of Scotland; but throughout the other parts of the regions above specified it is more or less considerably diluted with other races; finally, it extends as a comparatively insignificant constituent of the population into eastern and southern Europe. In North America and other overseas regions, it is to be found in proportions varying with the place of origin of the white settlers. The characters of the Nordic race are the following (see plates). The stature is considerable, that of Nordic males being on the average 1·73 m., and this tallness being associated with great length of the limbs. The cranium is long and narrow, and the occiput projects markedly. The face is likewise long and narrow; the nose is prominent, slender, with a high bridge, sometimes straight in profile and sometimes showing a slight eminence where the bones pass into the cartilages of the nose. The chin is strongly marked; the cheeks are narrow and the cheek-bones are not prominent; the hair, soft, straight, or slightly waved, is fair or reddish; the eyes are light in colour, ranging

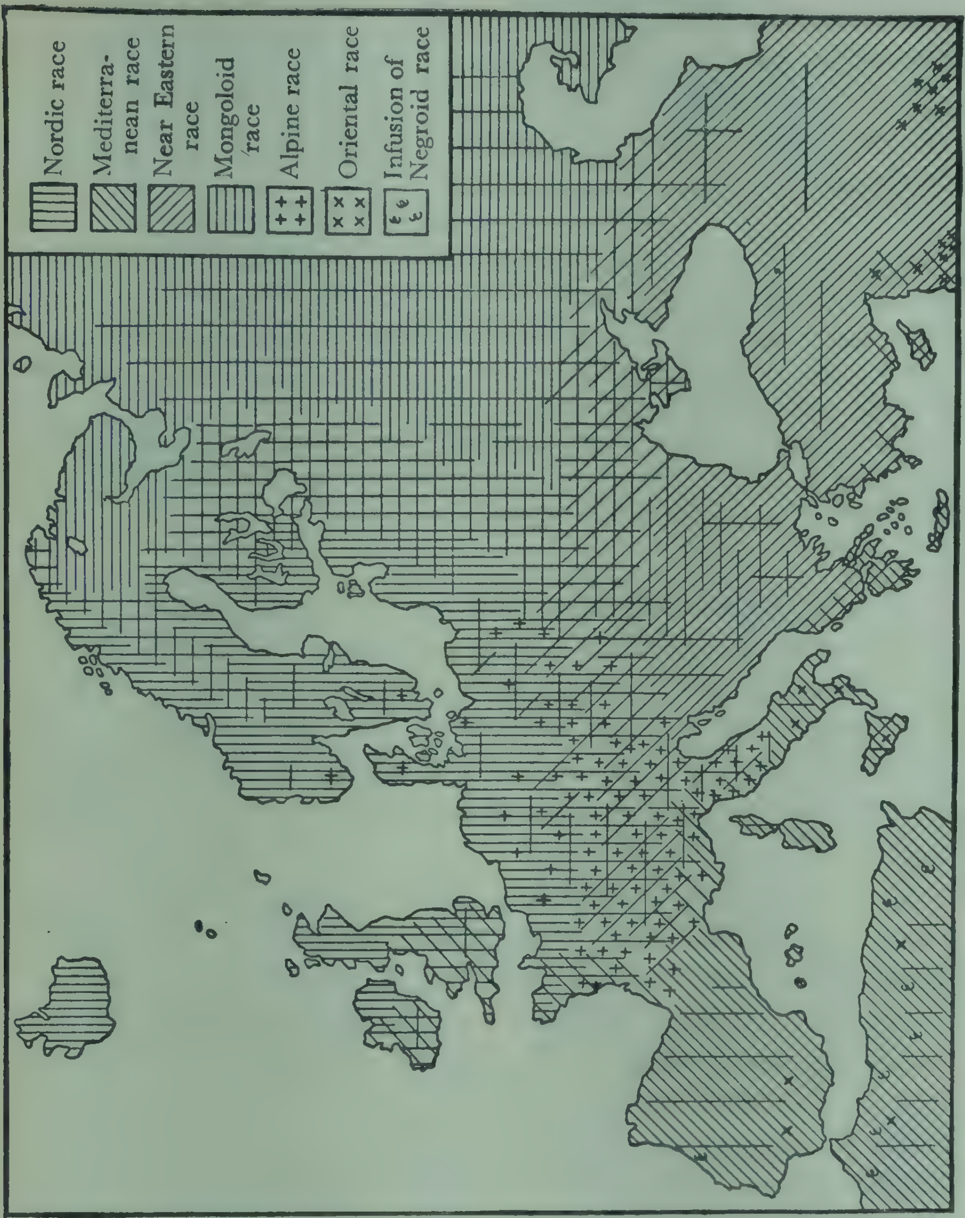

Nordic race
Mediterranean race
Near Eastern race
Mongoloid race
Alpine race
Oriental race
Infusion of Negroid race

FIG. 31.

Racial map of Europe.

The map shows how Eugen Fischer and Fritz Lenz, the compilers, contemplate the distribution of the races of Europe. The direction of the lines (not their density) indicates the various racial elements. For instance, the Nordic race is indicated by lines running north and south ("coming from the north"); the Mongoloids are shown by lines running east and west ("coming from the east"); and so on. In this way the origin of the respective races is graphically shown, as well as their present distribution. The density of the lines indicates the extent to which a race predominates in a particular area; when the lines are far apart, the race they indicate constitutes a smaller proportion of the population. The Dinaric race, being akin to the Near Eastern race, is indicated in the same way by lines running from south-east to north-west.

from blue to grey. The skin is fair, with little tendency to bronze in sunlight, so that it is extremely sensitive to the effects of the sun. Within the Nordic race, moderate local differences of breed can be made out, but these cannot be considered here in detail.

Typical Nordics, male and female, are the two young people shown in Plate I. The man, 24 years of age, whose father was a Low German and whose mother came from the Baltic region, is 183·9 cm. in height, dolicocephalic (index 78), blond (hair tint no. 26), with light greyish-blue eyes.

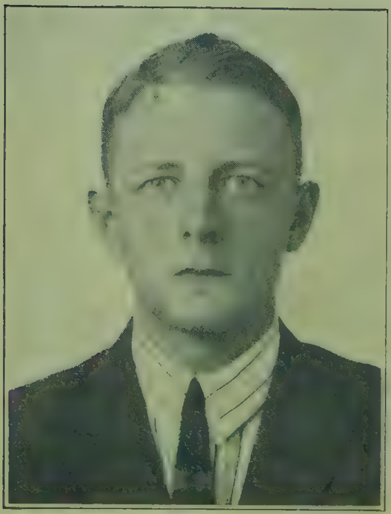
The young woman, from Hamburg and Hanover by parentage, 21 years of age, is 163·8 cm. in height, dolicocephalic (index 75), very fair (no. 17), and blue-eyed.

The Mediterranean race occupies the coastal regions of the Mediterranean. It forms the main, and in some parts the almost exclusive, constituent of the population of Spain and Portugal, the larger and smaller islands of the Mediterranean, and southern Italy almost as far north as Rome. Scattered elements of the same stock are found in the south of the Balkan peninsula and in Crete.

It occupies also the whole of the northern littoral of Africa from Egypt to Morocco (although, as will be shown below, there is considerable admixture of blood in these regions) ; and thence waves of it run inland, on the east up the Nile and on the west along the coast, where it certainly reaches the West African islands, although there it is strongly mixed. Mediterraneans are small in stature, the smallest of the Europeans. The average height of the males is 1·61 m. They have, like the Nordics, long and narrow crania, but smaller, and with a steeper forehead (see the plates). The face is small and oval ; the nose is straight, somewhat prominent, but smaller and a little wider than that of the Nordics ; often the fronto-nasal angle is remarkably flat (producing what is known as the Greek profile). The hair is of a very dark brown, being by non-experts usually described as "black". The eyes, too, are dark brown. The skin has an unmistakably brownish tinge, and is in addition slightly bronzed by the southern sun.

The Alpine race is scattered across a wide strip reaching from

PLATE I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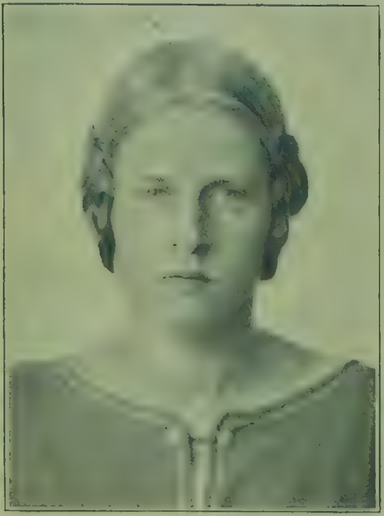
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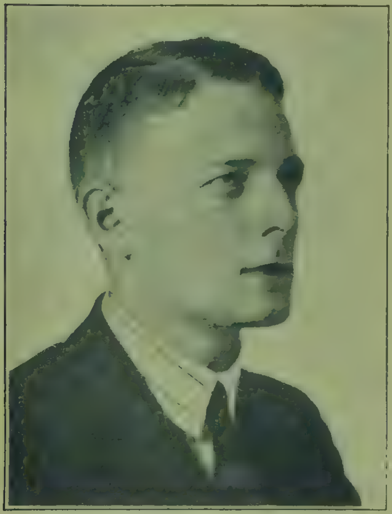
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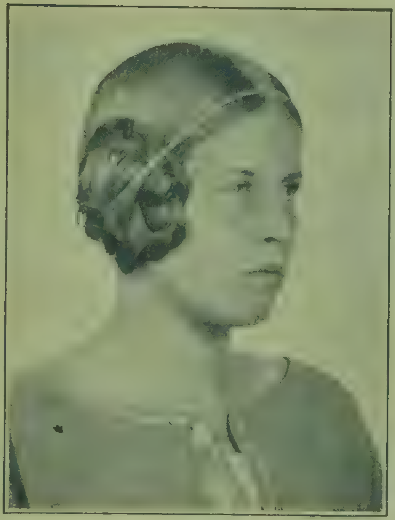
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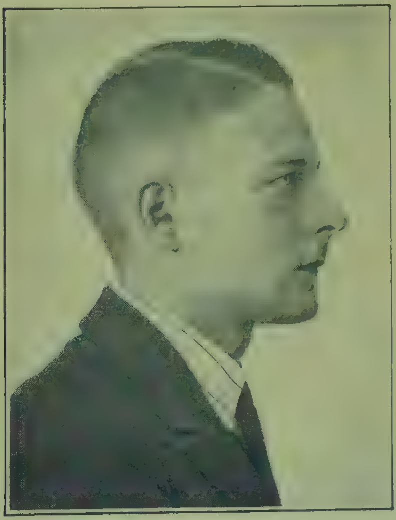
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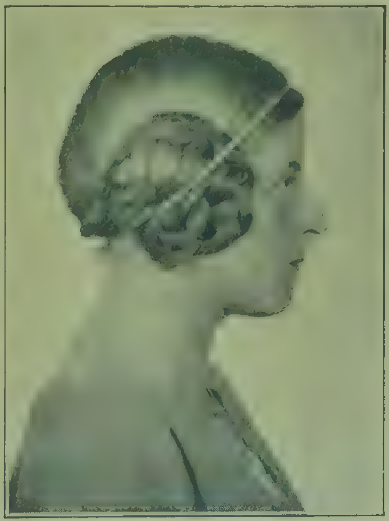
2c

east to west across the Alps of Central Europe.[1] It exists most free from admixture in south-western and central France from Gascony across Auvergne into Franche-Comté, and in the genuinely Alpine region, especially in its southern part, but thrusts out thence northward into the domain of the Nordics and southward into that of the Mediterraneans as far as Central Italy. From Tyrol and the eastern Alps, an offshoot of the Alpine race, containing much admixture, spreads northward and north-eastward into Central Germany; but, owing to the lack of anthropological data, the eastward boundary of its extent cannot be determined. Likewise it is still impossible to-day to delimit its extent south-eastward as against the domain of the Dinaric race. The characteristic features of this Alpine race are as follows (see plates). The stature is moderate, being on the average 1·63 to 1·64 m. in males; the cranium is round, the index as determined in living subjects ranging on the average from 85 to 87; the forehead is upright; the frontal and parietal eminences are rather strongly marked; the occiput is moderately curved, but not markedly prominent. The face is broad and rounded; the chin is pointed; the nose is coarser, shorter, and broader than in the other races, and in profile is slightly concave. The hair and the eyes are of a medium brown tint, the skin not being so light in colour as in Nordics, but lighter than in Mediterraneans, and it tends to assume an equable brown tint through the effect of sunlight.

Finally we come to the Dinaric race.[2] This race mainly inhabits the Balkan countries, being thickest in Bosnia and Herzegovina, and next to this in Croatia, Slavonia, and Albania. It spreads to the southernmost point of the Balkan peninsula, although in Morea the Mediterranean race is likewise strongly represented. To the south-east, the race spreads across the straits into Asia Minor, and can here in the light of our present knowledge scarcely be marked off from the "Near Eastern race" (see below). North-westward, it radiates into the Alpine region; and Dinaric elements spread across the Austrian highlands far into South and even Central Germany. The type cannot be mistaken. The hair and the eyebrows are so dark as to be almost black, the hair is straight, and there is a strong growth of hair on the face. The head is short and high, not very wide, so that the cranial index is a medium one, ranging from 81 to 86 (see plates). Especially characteristic is the extreme flattening of the occiput, so that the back of the head when seen sideways looks as if it had been shaped vertically with a chopper. The face is long, rough-hewn, while the nose is large, prominent, and often conspicuously hooked. Hardly any other race has so strongly developed a nose. The aquiline type of nose and the sharply curved hawk-like nose are about equally represented. The stature is considerable, that of the males ranging from 1·68 to 1·72 m. These four racial types, very variously intermingled, comprise the main bulk of the European populations. In addition, however, there are various extraneous constituents, appearing here and there in diverse proportions. From the east, in especial, Mongoloids have made their way into Europe; from the south-west have come people of Arab stock; and all over Europe there are Jews.

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[1] Günther denotes this race by the misleading name of "ostisch". (See map, p. 187.)

[2] So called after the Dinaric Alps, the general name given to those mountain ranges in Dalmatia, Bosnia, Herzegovina, and Croatia which are clearly a continuation of the main Alpine system.

The next problem that comes up for consideration is that concerning the origin of the European races. There is, as yet, no evidence of the persistence of the earliest human inhabitants of Europe (the Neandertal man, etc.). There is, however, some warrant for the view that the Cro-Magnon stock belonging to the close of the paleolithic period, formed part of the ancestry of contemporary Europeans. At any rate there seems to be some connexion between the Cro-Magnons, on the one hand, and the Mediterranean and Nordic races on the other. But it remains a moot point whether the Cro-Magnon race sprang from the earlier race which must have also given rise to the Mediterranean race (we have as yet no fossil evidence as to the existence of such a race, and the Cro-Magnons are certainly of a bulkier type than the Mediterraneans); or whether the contemporary Mediterranean form may have directly evolved out of the Cro-Magnon type. (There are obvious relations between the Cro-Magnon skull, skulls from Teneriffe and Egypt, and, above all, certain neolithic human remains discovered at El Argar in Spain.)[1]

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[1] Scheidt, Rassen der jüngeren Steinzeit in Europa, Munich, 1924.

At any rate we are entitled to suppose that the Mediterranean race was at the close of the paleolithic period already occupying much the same habitat as to-day. On the other hand, the anatomical characters of the Cro-Magnon skeletons indicate racial relationships with the megalithic population of Europe—with the dolmen builders of the neolithic period who dwelt along the Scandinavian, Danish, and other coasts. It seems probable that the makers of the megalithic monuments were descendants of the Cro-Magnons. If so, however, they must have undergone considerable modifications in form. Especially, the shape of the face must have changed a great deal, from being broad and low to become long and narrow. But the broad Cro-Magnon type of face still turns up occasionally to-day. The power of inheritance is continually reproducing it anew. Thus, side by side with the Nordic forms in the population (developed out of the Cro-Magnon race), there are still to be encountered to-day, practically unchanged, broad-faced types, such as may be seen at times alike in Lower Saxony and in Sweden. For this reason, it seems to me that we have no warrant for assuming the Nordic race to have had a quite peculiar origin, from an unknown root.[1] We may, however, suppose that when the great ice-sheet receded from northern Europe, man made his way northward following the reindeer herds in their secular migration, and that a group of human beings isolated in the north became transformed into the Nordic race. It may have been quite a small group which, under stress of rapidly changing conditions (climate, beasts of the chase), was exposed to exceptionally rigorous selection and was persistently inbred, thus acquiring the peculiar characters which persist to-day as the exclusive heritage of the Nordic race. Inasmuch as previously, owing to the presence of the ice-sheet, there can have been no settled population in our hypothetical region, the Nordic race thus developing would have been purely indigenous. Pretty soon, however, there arrived from the east a second wave of immigrants, the ancestors of the contemporary Lapps. From the very first, among the long and narrow skulls of the dolmen builders and among the human remains of all subsequent prehistoric periods, we find a sprinkling of round crania—just as we find them to-day, a sprinkling of the round skulls of the Lapps among the long skulls of the Swedes. Then, along the southern border of the primitive distribution of the Nordic race, there must also even in prehistoric times have been an admixture of Alpines, whose skulls are to be found as the so-called Borreby type in Denmark and in Holland. From the east, further, even as early as this must have come forerunners of the Mongoloid invasion. Indeed, the question arises whether the

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[1] Paudler, Die hellfarbigen Rassen, etc., Heidelberg, 1924.—Hauschild. op. cit.

Alpine race itself may not have been the westernmost offshoot of the Mongoloids. It is certainly an error to suppose that in the earlier Stone Age the Mongoloids had already advanced westward as far as the Atlantic Ocean; but we are perhaps within our rights in the belief that at the time when the Mongoloids were being separated from the Europoids, and before either stock had fully developed its distinctive characteristics, a westernmost group of the commencing Mongoloids became segregated. That would explain why members of the Alpine race exhibit certain Mongoloid characters (shape of cranium), while being devoid of the Mongoloid spot and of the Mongoloid peculiarities of the eyelids and the palpebral fissure, etc. On the other hand, the Mongoloid forerunners in the north-west, the Lapps, have these unmistakable Mongoloid traits, and must therefore have migrated after the Mongoloids had developed their full racial characters. From the first, not yet entirely specialised Mongoloid root, developed the Alpines, who, already dwelling in Europe during the Ice Age, thrust forward to the north into Jutland, to the north-west into Holland, and perhaps made their way into England—while to the south we find unmistakable vestiges of them in Spain and Portugal.[1]

Chekanowsky and his pupils, discovering a frequent correlation of blondness and brachycephaly in Poland, infer that a white race came into being in Poland and eastward of the Baltic. Günther, who supports this view, speaks of the hypothetical race as the "East-Baltics". The theory is dubious. A combination of the before-mentioned characters might well have arisen as a result of crossing.

Finally the Dinaric race must, so far as we know, have come to Europe much later. There is no evidence of its existence in Europe during the Ice Age. In those days, we must suppose, it was to be found only in Asia Minor. The first traces of its appearance in Europe date from the neolithic period, and it would seem to have undergone fairly extensive diffusion during the Bronze Age. In the neolithic period, the most advanced outposts of the Dinaric invasion may have left their vestiges in the round barrows of the British Isles. Consequently there is no reason to be surprised when to-day we encounter Dinaric racial characters in the very heart of Europe. In like manner, Dinaric elements would seem to have passed from Asia Minor or from south-eastern Europe, by way of Crete, westward into Sicily and southward into Egypt.

During the paleolithic period, the Negro population of Africa also crossed to the southern shores of Europe, as is proved by the

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[1] That is why Günther's name of "ostisch" for this race is misleading.—Regarding Spain, see Scheidt, Rassen der jüngeren Steinzeit, p. 90; and, above all, Obermaier, El hombre fossil, second edition, Madrid, 1925.

PLATE II

RACIAL TYPES

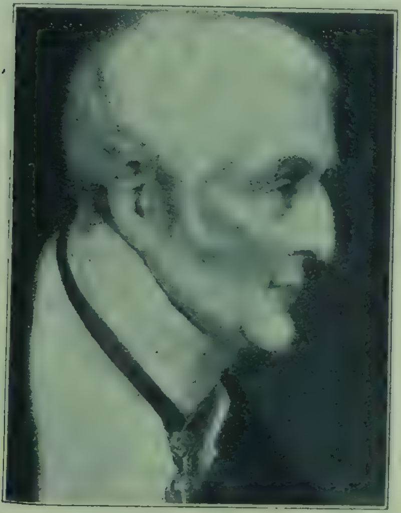
1

North Germany (the hair is rather too dark).

2

Sweden.

3

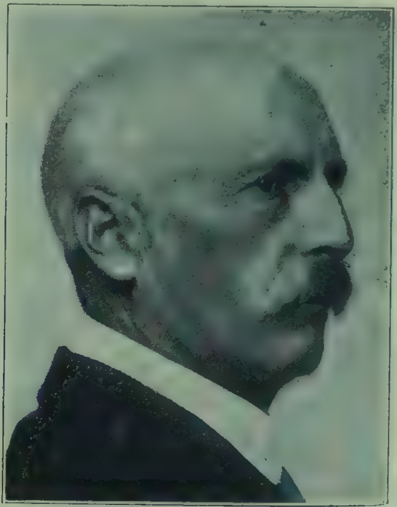


England (note the strongly arched nose).

4

Flanders.

5



North Germany.

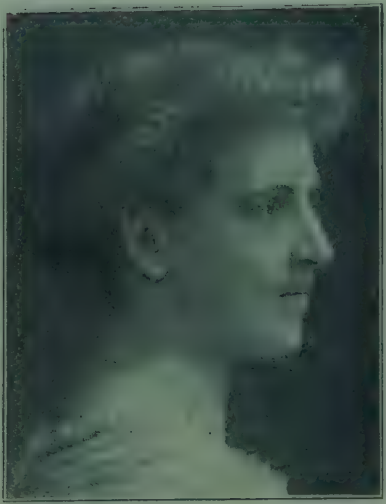
6

Hanover.

Nordic Race.

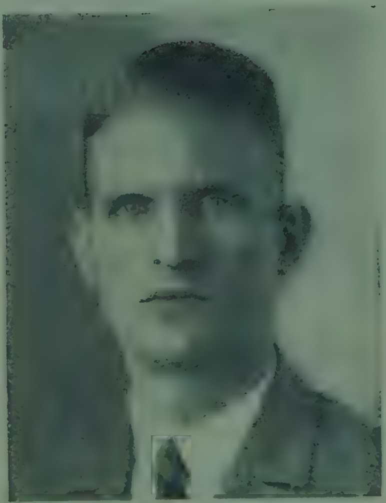
PLATE III

7



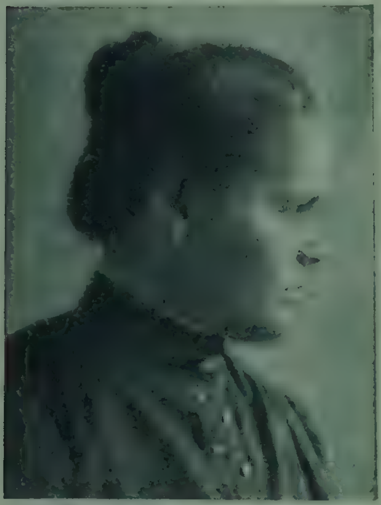
Nordic Race (Central Germany).

8



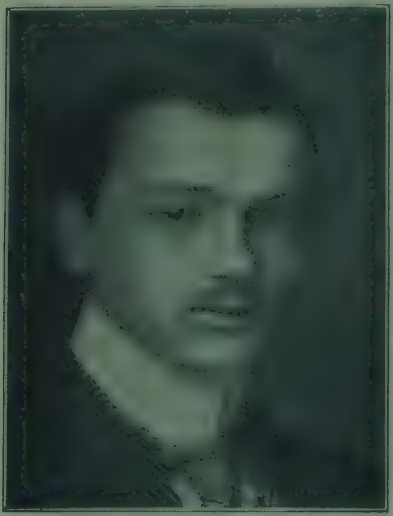
Nordic Race (Mecklenburg).

9



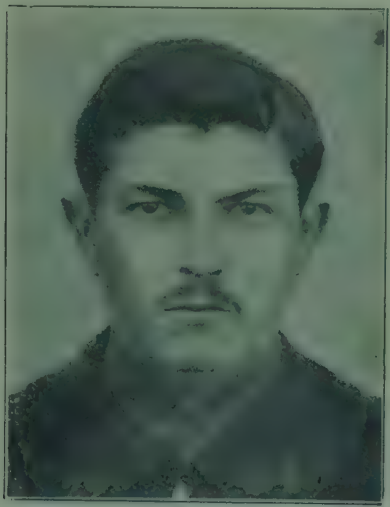
Alpine Race (Southern Black Forest).

10



Alpine Race (Dresden).

11



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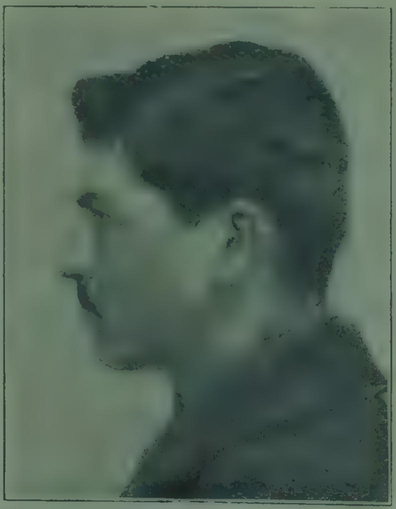


Alpine Race (Maritime Alps).

13

Alpine Race with slight admixture of Nordic (Central Germany).

14

Alpine Race with some admixture of Nordic (Central Baden).

15

Mediterranean Race (Southern France).

16

Mediterranean Race (Upper-class Portuguese in Brazil).

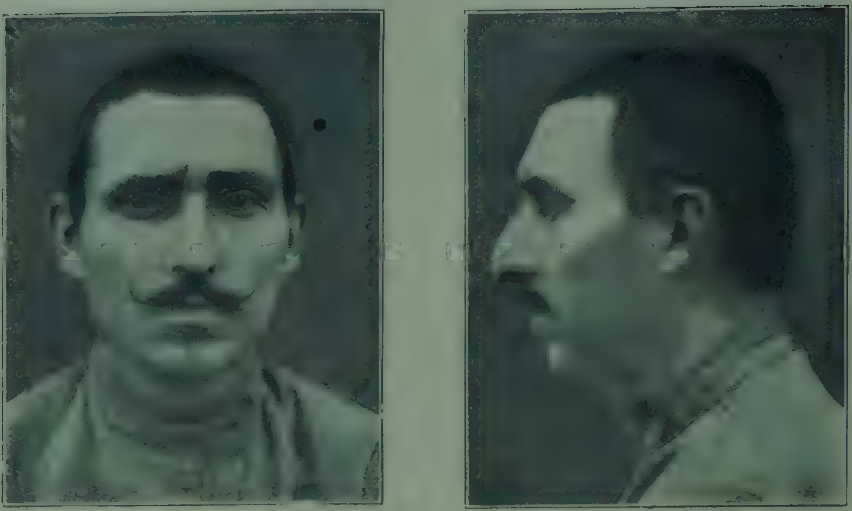
17

Mediterranean Race with some admixture of Nordic (Western France, Brittany).

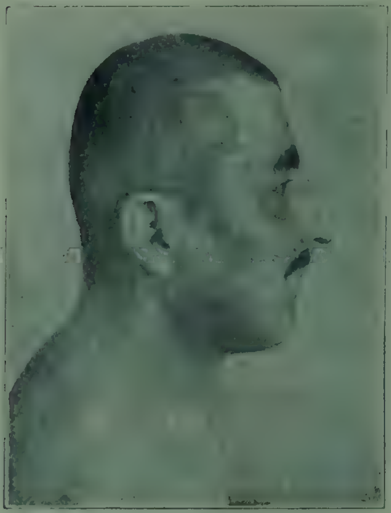
18

Mediterranean Race (Eastern Pyrenees).

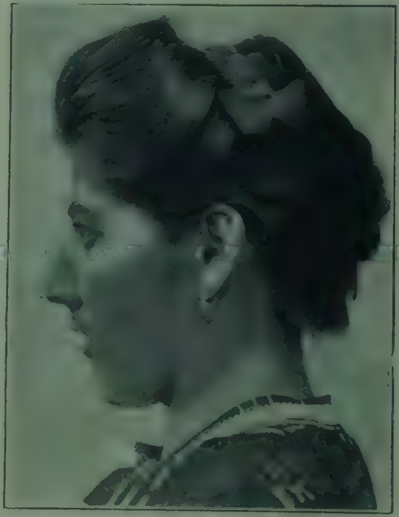
PLATE V

19  20

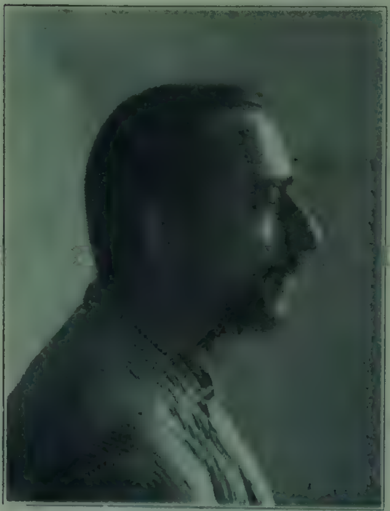
Near Eastern Race (Bessarabia).

21 

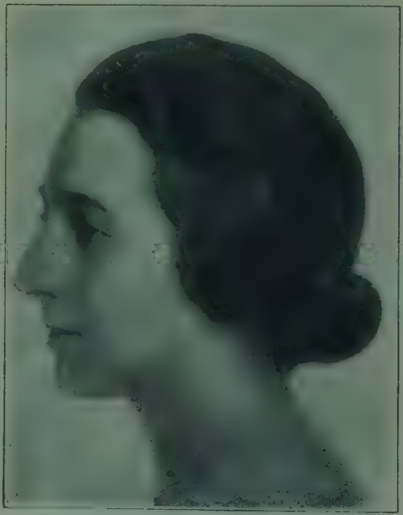
Near Eastern Race (Armenia).

 22

Dinaric Race with a little Nordic admixture (Tyrol).

23 

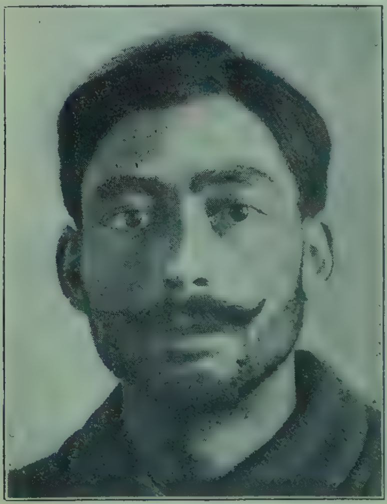
Dinaric Race (Southern Baden).

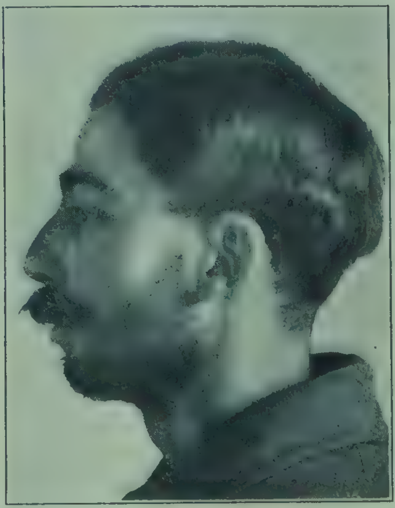
 24

Oriental Race with Near Eastern admixture (Belgian Jewess).

PLATE VI

25

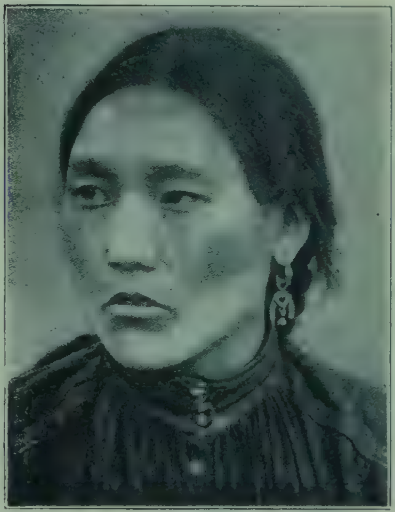


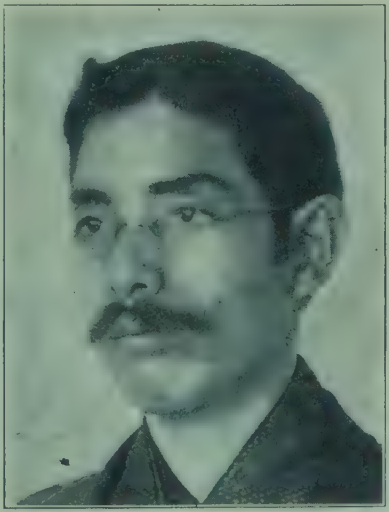


26

Oriental Race (Algeria).

27

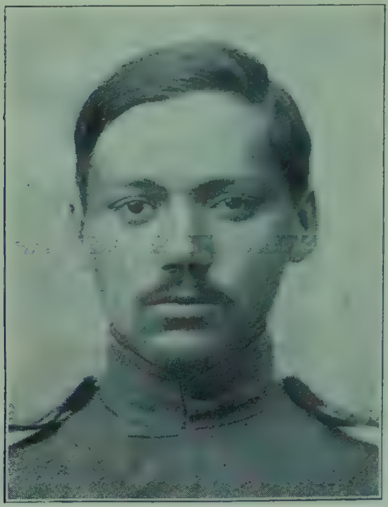


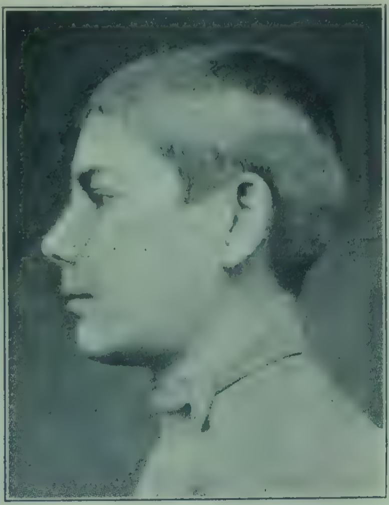


28

Mongoloid Race (Yakutsk Woman). Mongoloid Race (Upper-class Japanese).

29



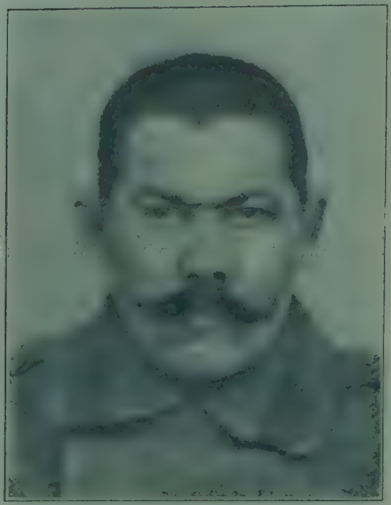


30

Mixed Mongoloid and Nordic
(Latvia).

Mixed Mongoloid and Nordic
(Kursk in Central Russia).

PLATE VII

31


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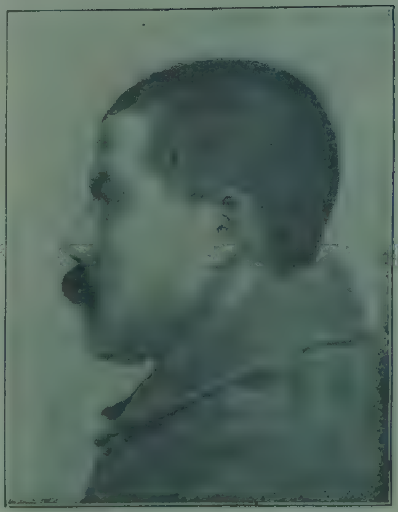
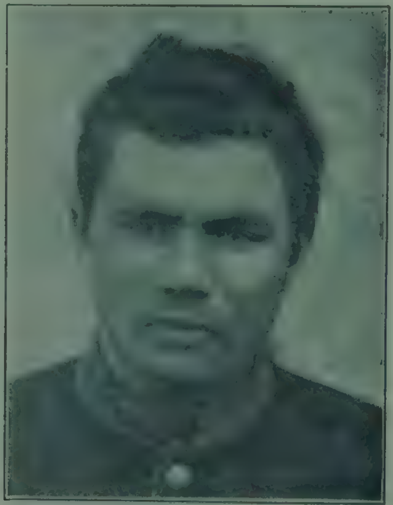


Mongoloid Race with a little European admixture (Ryazan in Central Russia).

33


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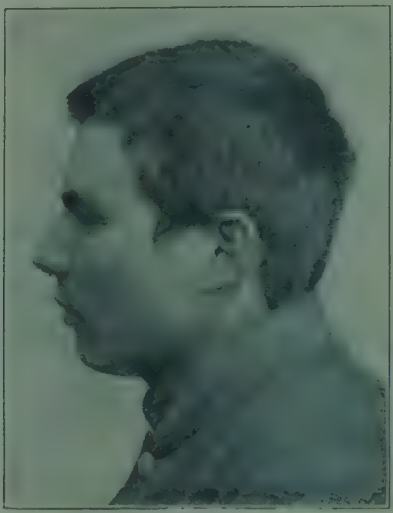
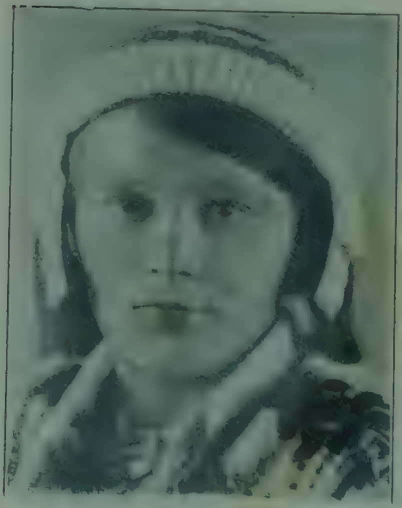


Mongoloid Race with considerable Nordic admixture
(Saratov in South-East Russia).

35


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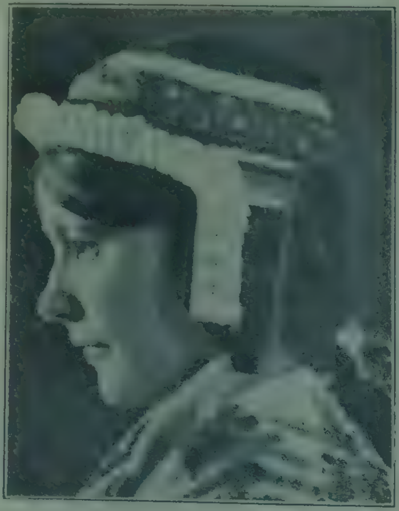
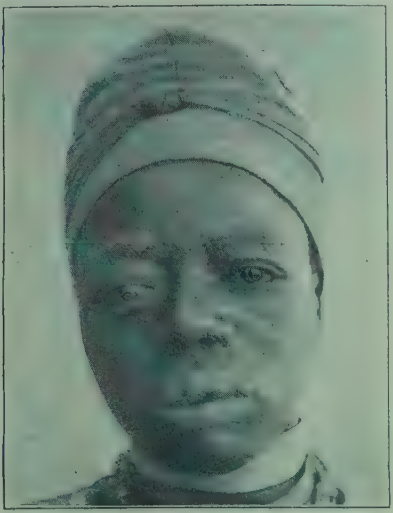


Mixed Mongoloid and Nordic (Lapland).

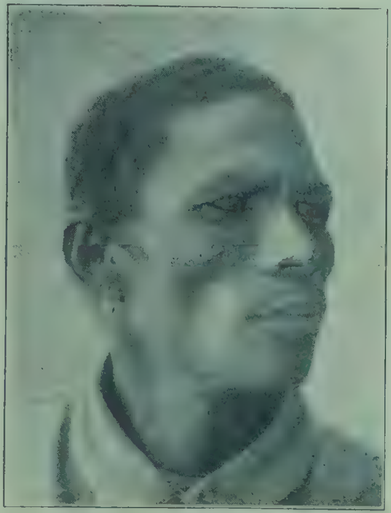
PLATE VIII

37



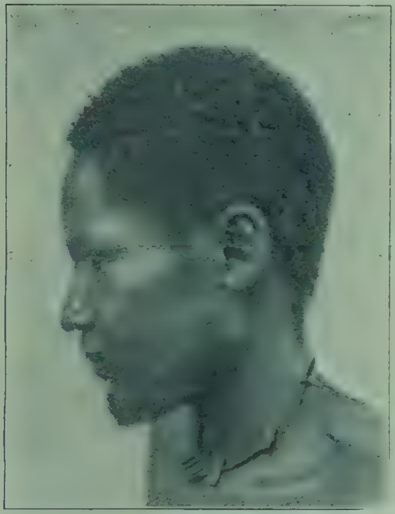
Negroid Race. A Herero woman from German South-West Africa.

38



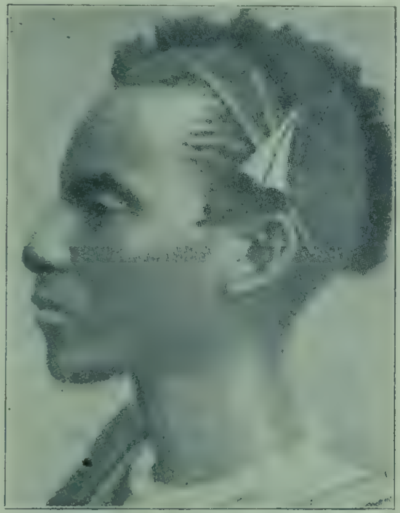
Negroid with a little Oriental admixture (Herero man).

39



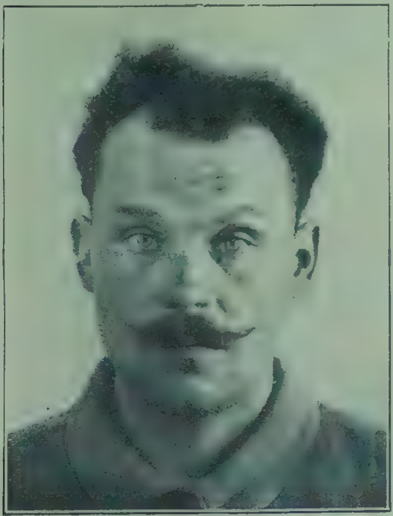
Negroid with considerable Oriental admixture. An Ovambo man from German South-West Africa.

40



Oriental with considerable Negroid admixture. A Hima man from Ruanda, in East Africa.

41



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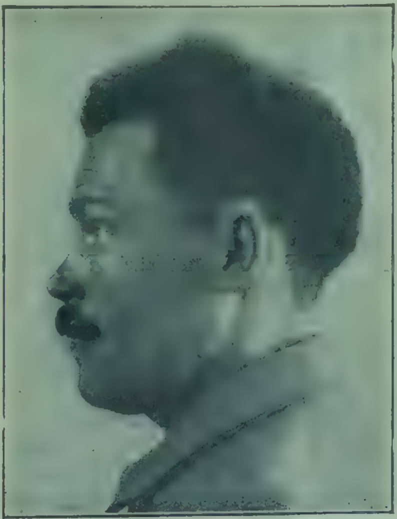
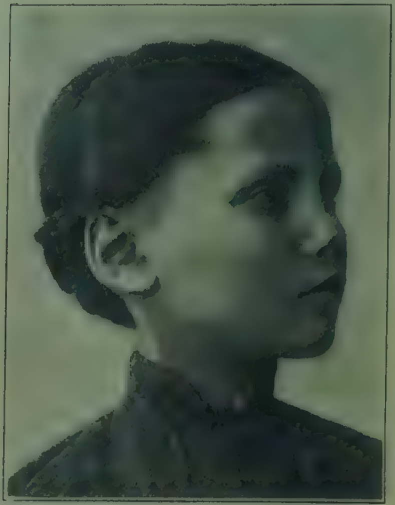


European of Mixed Type with presumably some Negroid admixture (the hair is Negroid) (Mohilev, in West Russia).

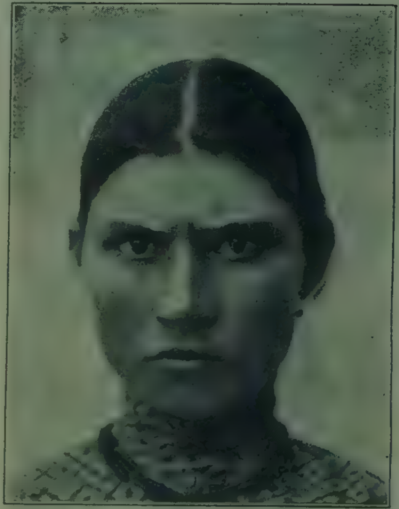
PLATE IX

43



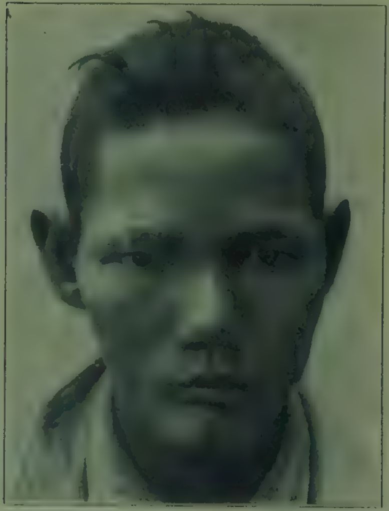
Half-breed girl whose father was European and whose mother was Hottentot.

44



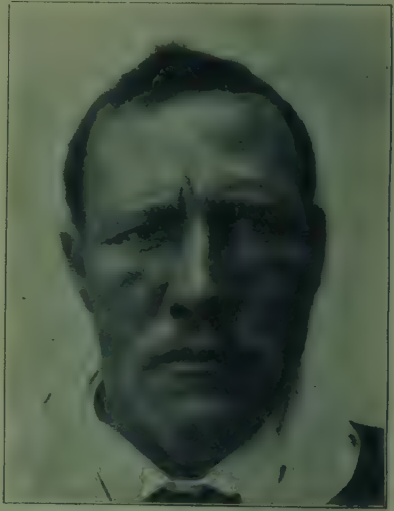
Woman born from the recrossing of a half-caste Hottentot woman with a European.

45



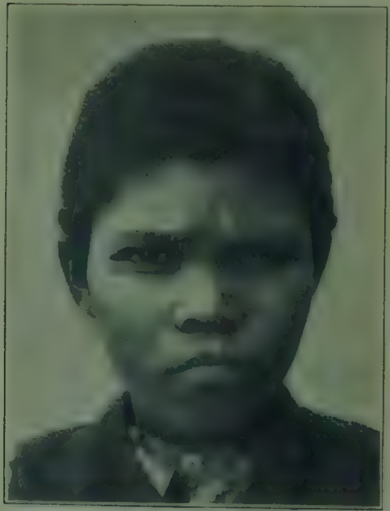
Mixed-breed man of the third generation having rather more Hottentot than European blood.

46



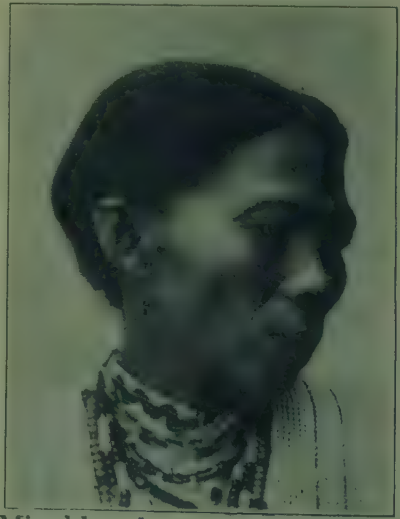
Mixed-breed man of the fifth generation having rather more European than Hottentot blood.

47



Mixed-breed boy of the fourth generation having rather more Hottentot than European blood.

48



Mixed-breed woman having about equal amounts of Hottentot and European blood.

Cross-Breeds between Europeans and Hottentots in German South-West Africa.

discoveries made in the Grimaldi caves; but these Negro elements have not entered to any extent worth considering into the composition of the latter-day European population.

Now that we have sketched the origin of the main European races, we must briefly consider the connecting-links between these first beginnings and contemporary conditions. Although at the close of the Ice Age the three chief European races were already settled in much the same habitats as they occupy today, the evidence we possess concerning numerous subsequent migrations throws a good deal of light upon the intermingling of the extant peoples. What is known in history as the period of folk migrations was certainly no more than the last phase, and was probably not the most important phase, of extensive migrations during prehistoric times. Before the migration of the Teutons, there were the migrations of the Celts and the Slavs; and before any of these there were the prehistoric migrations which must have gone on throughout the neolithic period, shuffling the various peoples all over Europe. We cannot, as yet, invariably trace a harmonious relationship between cultural trends and anthropological types, but here and there we can unmistakably detect racial displacements which accord with the folk movements. In the eastern part of central Europe (in Bohemia, for instance, and in Silesia) there were in neolithic times round-skulled men (perhaps of the Alpine race) who were subsequently displaced by longheads. In the tombs of these latter, the great majority of the skulls of males are dolicocephalic, whereas many of the skulls of females are brachycephalic, so that here we have the typical picture of the conquest of a settled population by invaders.[1] During this same neolithic period, the pile-builders or lake-dwellers of central Europe were of the Alpine race; and at about the same time and in the same region there existed a nomadic immigrant population whose type of civilisation is characterised by the making of the so-called bell-goblets, and the skulls of these people are typical of the Dinaric race.[2] Here, also, we have evidence of migrations and of shiftings of population. It is likely enough that the developments of the subsequent Bronze Age may have been determined by such shiftings. Much easier to read is the record of the skeletal remains belonging to the days of the Celtic and Teutonic migrations, for the typical tombs of the Celts, the Teutons, and the Slavs, respectively, contain the bones of the inhabitants of Europe at a time approximating to the commencement of the Christian era. In the unmistakably Teutonic graves belonging to the period of the folk migrations—in the graves of the Franks, the Saxons, and the Alemanni—all the skulls are of a purely Nordic racial type (identical with that found in Sweden to-day), no matter whether the disinterment has taken place in South Germany, France, or Italy.

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[1] Reche, Zur Anthropologie der jüngeren Steinzeit, etc., "Arch. Anthr.", 1908.

[2] Schliz, Die vorgeschichtlichen Schädeltypen, etc., "Arch. Anthr.", 1908, 1910, 1914, etc.

The question as to the original home of the Indo-Germans is closely connected with the problem of these prehistoric folk migrations. Philological, archaeological, and anthropological researches combine to indicate that the primal home of the Indo-Germanic languages must have been in northern Europe. To our purpose it is of no moment whether this may have been in Scandinavia, in the plain of North Germany, or on the shores of the Baltic. Whatever the precise locality, somewhere in northern Europe there must have dwelt the primal Nordics from the days when the northward retreat of the ice made human settlement of these regions possible. These Nordics were the sustainers of the original Indo-Germanic language and civilisation; and whithersoever Indo-Germanic tongues were born in the migrations that took place after the severance into distinct stocks, the carriers were individuals of Nordic race, or of mixed Nordic and alien blood. The last of the great waves to spread from the original home, that of the Celts, that of the Teutons, and that of the Slavs, were primarily not distinguished one from another in respect of race. Their destinies, however, were notably different. The Celtic stream, insofar as it remained in Europe, flowed partly into the region of the Mediterraneans (Spain and Italy), partly into the region of the Alpines (central Europe, France, South Germany, Switzerland, North Italy, etc.), partly into the region of the Nordics (England), and partly into the region of the Dinarics (the Balkans). In the two first-named regions of mingling, the Nordic type was obscured by the selective processes above described, being in central Europe fused with the Alpines, and in southern Europe absorbed by the Mediterraneans. But the Nordic culture was preserved. The new sustainers of this Celtic culture in central and western Europe were now for the most part dark-haired, and the Teutons who subsequently arrived found in southern France (for instance) a multitude of dark-haired "Celts". Even to-day, the non-expert in these matters is generally inclined to think of the Celts as dark-haired and to regard the extant dark-haired elements in our population as of Celtic origin, whereas in reality their origins go back to the Alpine race of the neolithic or even of the paleolithic age. The eastern stream, the Slavic, became more mixed racially than any of the others. Its mission was to safeguard Teutonism from the Mongolian flood—a role in universal history that was inconspicuous, perhaps, but was in truth of overwhelming importance. Like a living wall the primitive Slavs, tall and fair-haired, pushed eastward in front of their Teutonic brethren and absorbed into themselves the Mongolian invasion in successive waves. Thus it was that—filtered, as one might say, mitigated, diluted—with the subsequent return of the Slav populations from east to west, Mongolian racial elements were introduced into the heart of Europe, far into the domain of Teutonism, up to and even beyond the Elbe. Hence we find to-day certain Mongoloid characters as elements in what is mainly the Nordic-Alpine compost of the German people; but the Mongolian admixture would be far more extensive and would have had much more influence upon the whole had not the Slavic wall been a barrier against an overwhelming influx. What is so often described as a Slavic type, characterised by broad faces, rather prominent cheekbones, and squat figures, is in reality a Nordic-Mongoloid fusion. Everywhere the contemporary Slavs have a considerable Mongolian ingredient in their composition, and among the Southern Slavs considerable Alpine and Dinaric racial elements are also intermingled. The Teutonic stream, finally, remained nearest to its original home, and was least influenced by the admixture of foreign races. In South Germany, indeed, there was a considerable infusion of Alpine elements; in Scandinavia, Mongoloid ingredients were introduced by the Lapps; and in

North Germany, there came, as just described, a late introduction of Slav elements. Throughout north-western Germany, Holland, Belgium, and northern France, the Nordic race is predominant throughout wide areas. In Belgium and northern France, however, there has been a gradual dilution with the Alpine stock. Where the Teutonic wave broke on foreign shores, at an increasing distance from the primitive Nordic home, the Nordics were absorbed into the other races, and for the most part disappeared. Think in this connexion of the Burgundians, the Goths, the Vandals, etc. But even to-day, in the countries of southern Europe we find many more fair-haired persons than we find of dark-brown-haired persons in Scandinavia, although the brunette trend is dominant in inheritance, so that whereas no blond northern Europeans conceal the brunette factor, a great many dark southern Europeans carry a hidden blond factor. Herein we discern a plain indication that racially as well as in other ways, southern Europe has been greatly influenced by Nordic immigration. In Great Britain, along the southern coastal regions, there has been a considerable infusion of Mediterranean elements among the Nordics, although elsewhere Nordics predominate throughout. There can have been very little introduction of Alpine and Dinaric elements into Great Britain and Ireland.

Thus all the races of contemporary Europe are compounded out of the same racial elements in the main, the differences between them depending upon the proportion of the various components and upon the extent to which entirely alien elements have been introduced. Among such alien elements, the Mongoloid has already been mentioned. As far as the Spanish population is concerned, reference must be made to the marked traces of admixture left by the Arab invasion and by the centuries of Arab rule, so that in all parts of the peninsula we can still plainly discern evidence of the admixture of Oriental elements (see below) and here and there likewise of Negroid constituents. Other migrations recorded in history are still producing their effects to-day, such as the settlement of the Mongoloid Magyars in Hungary, of the Mongoloid Bulgars and Turks in the Balkan peninsula, the invasion of the Huns, etc. Finally, we have to remember that the extensive freedom of intercourse throughout the centuries (a freedom which is still more marked to-day) has led in thousands of individual cases to the introduction of alien racial elements. Isolated Negroes, Mongols, etc., have been imported into Europe. Southern Europeans have removed to central and northern Europe, and conversely. At the present time, it is the Jewish admixture which is playing the most extensive part in this connexion, for it introduces a considerable amount of the Near Eastern racial element (see below). All these comings and goings help to explain, in the light of the known laws of heredity, the peculiar traits which sometimes appear in particular families and individuals. The introduction of hundreds of thousands of prisoners of war from the most various countries of the world into Germany, the long-continued presence of the coloured troops in the occupied territories, and above all the greatly increased settlement of such alien elements in France since the close of the war, will not fail to have plain effects in the future!

There can be no doubt that the mental differences between the various European nations are in great measure dependent upon these racial differences. Obviously the four great European races, and the extra-European races which have contributed to the European compost, all have mental heredity factors peculiar to themselves. The processes of crossing and selection, varying from nation to nation, will have had specific effects in each case, determining the mental disposition of the nations concerned. Nor can we suppose that this process has now come to an end. Under our own eyes selection is still going on, with varying intensity, in relation to the various racial components of a nation, just as it has gone on for hundreds and for thousands of years, so that the composition of each people is to-day being modified and its destiny is being in part determined by this unceasing process.

In conclusion, let us glance at the European population of the transoceanic countries, and especially of the United States of North America. There we see kindred minglings to those with which we are already familiar in Europe. Furthermore, in many of the States, especially in the South and in the Middle West, there is a strong infusion of Negro blood, whose effects are manifest in the cultural development of these regions. As regards the racial composition of the North American population, it is obviously of great importance that to begin with the main source of immigration was Britain; that then the chief influx came from Germany; and that of late decades increasing proportions of the immigrant waves have been derived from southern and eastern Europe. Notwithstanding the persistence to-day of considerable social differences between the populations derived from these various sources, in days to come they will freely mingle. As this goes on, social selection (and perhaps natural selection) will play a considerable part in the result, above all in the matter of great differences in the rate of multiplication of the various elements. Hence no valid prophecy can be made to-day as to the development of this population, and the only thing we can say with certainty is that the present proportional composition of the various stocks can give no certain clue as to what will be the character of the compost in a few generations. Moreover, after all that has been said above, it can hardly be needful to insist that an immigrant described as of "German" or of "Italian" origin may be racially of a very mixed type.

Similar considerations apply to the other great areas of European settlement across the sea—to Australia, South Africa, etc.

I need hardly repeat that the racial changes which will occur in the immigrant population of these countries cannot fail to influence their history.

In the United States, advantage was taken of the world war to conduct an extensive anthropological investigation, relating to more than two million men enrolled in the army.[1] The mean stature (of white and coloured taken together) was 171·99 cm. This shows no notable change from the results of a statistical inquiry made fifty years earlier during the civil war. The colour of the eyes was in 35·8 % blue, in 26·5 % blue with modifications, in 26·1 % light brown, in 11·6 % dark brown. The high percentage of blue eyes is remarkable, seeing that the aggregate material included coloured persons! We do not know, however, to what extent selection (disinclination for war service, varying fitness of the different nationalities) may have influenced the composition of the material. In the civil war, 45 % of the soldiers had blue eyes, so that there has been a notable decline in the percentage (the earlier material included no coloured persons, and no whites from the Southern States). As to the colour of the hair, in 4·7 % it was fair, in 22·3 % light brown, in 21·3 % medium brown, in 46·1 % dark brown, in 1·3 % red, in 4·5 % dark red.

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[1] C. B. Davenport and G. Love, Army Anthropology, being vol. XV of The Medical Department of the U.S. Army in the World War, Washington, 1921.

The work contains an enormous mass of anthropological data, including such as relate to the individual national constituents and to the various localities of the Union; also extensive statistics concerning the frequency of various diseases and errors of development. Unfortunately, I cannot attempt to do justice to these matters here.

Before leaving the subject, I will make a brief reference to conditions in Hindustan.[1] In the first half of the second millennium before Christ, the "Aryan" Hindus were settled in the Punjab as a pastoral people. Somewhere about 1400 B.C. they advanced southward, in part driving the indigenous Dravidian population before them, and in part by degrees fusing with these to form one people into which elements of a more primitive population (the Veddah race, see below) were absorbed. The language and the culture of the Hindus are Indo-Germanic. In growth, shape of cranium, shape of nose, and tint of skin, they disclose a Nordic origin. It is true that the skin is no longer "white", there having been intensive mixture with a darker stock, and the selective elimination of those with fair skins who were inadequately protected against the tropical sun having been so rigorous that the pure homozygotic blonds have been practically eliminated. The upper castes (the Brahmans) exhibit the Nordic characters most clearly. It has been said, and with good reason, that in Hindustan an individual's social position is in inverse ratio to the breadth of his nose! Here in India we can see very clearly how racial admixture and selection work, how the shape of the nose, the length of the skull, and the stature, have maintained themselves, while the fair tint of skin has been eliminated. Thus some of the characters of the Nordic race, but not all of them, have been preserved—and there has been a persistence of the elements of that notable civilisation and of those artistic achievements which the Nordics created and have everywhere sustained.

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[1] E. Schmidt, Zur Anthr. Sudindien, "Arch. Anthr.", 1910.—Eickstedt, Rassenelemente der Sikh, "Zeitschr. Ethnol.", 1920–1921.

The Gypsies have certainly Indian elements in them, intercrossed with very various European ingredients.

2. ASIA MINOR

Prehistoric and anthropological researches make it fairly certain that long prior to the second millennium before Christ there was diffused throughout Asia Minor, Armenia, and Mesopotamia a population to which we may give the name of the Near Eastern race. It is very closely akin to the above-described Dinaric race, being distinguished from it only by a smaller stature. Presumably the two are sister races. All over the Near East, this "Near Eastern" race is still to be found in a fairly pure condition wherever it has been able to protect itself from the storms of the folk migrations of the last four thousand years. Such remnants are to be found in the remoter mountain districts of Asia Minor (for instance, the Allevi, the Kizilbashes, the Takhtaji, etc.), also among the Armenian highlands, in certain noble families, in sects, and so on.[1] Already in the early days, more than two thousand years before the opening of the Christian era, but at a time when the "Near Easterns" had long since split into considerable nations and had founded important realms (one of the most notable we now recognise to have been that of the Sibaraeans), they had been considerably influenced by immigrants from the north. Out of the shattered remnants of the Sibaraean kingdom, the Hittites (whose speech is now certainly known to have been Indo-Germanic) founded a new and powerful State. (The statues which used to be regarded as "Hittite" portraits, are now recognised to have been Sibaraean.)

The process repeated itself more than two thousand years later. Coming from the north-west across the Straits of Constantinople into Asia Minor, there was an Indo-Germanic irruption, and the newcomers had certainly a strong admixture of Nordic race. The Thracians of the Greek authors and the Amorites mentioned in the Bible were late manifestations of the same influx. Much more important, however, were the effects of an invasion from the south. During the first half of the second millennium before Christ, Semitic immigrants made their way upwards along the Euphrates and the Tigris. We learn of this folk movement in the biblical legends. In large measure the new arrivals imposed a Semitic culture upon the Near Eastern population, so that Semitic nations and States came into being and a mixed race was formed. The invaders themselves belonged to what I propose to call the "Oriental" race. The original home of this race is hard to ascertain. The Arabian peninsula (which in the diluvial period must have been fertile, just as was the Sahara at the same epoch) may well have served as the area in which the distinctive characters of this race were evolved; but it is just as likely that the "Orientals" came from the west. The Oriental race is distinguished by its small stature, by a long and narrow cranium, a narrow face with thick and fleshy lips, and a narrow, prominent, equably curved, and not excessively large nose. To picture it, think of the faces of the more refined Jews of southern Europe, as contrasted with the coarse features of the big-nosed Near Easterns. The Oriental race would seem to be closely akin to the Mediterranean, the two springing from the same root. The hair and the eyes of the Orientals are blackish brown, but the skin seems to have been exceptionally fair or to have become so. Such was the racial type of the original Semitic immigrants into the Near East. The "Semitic" peoples usually so-called, the Assyrians, the Babylonians, the Hebrews, and others, were formed in the main by a mingling of the Oriental and the Near Eastern races. But among these "Semites," just as among the chief races of Europe, foreign elements can be demonstrated in addition to the fundamental stocks. One of these is derived, as already said, from reiterated Nordic infusions; and there are also unmistakable though not very abundant Negroid components, which the Semites presumably acquired in Egypt. Finally, in the Sumerian monuments we see depicted types with small pointed noses. They may have been the outcome of an Alpine infusion; or perhaps of an admixture of Mongoloid blood from south-western Asia: but it is far more probable that they were offshoots of the Mediterranean race, which, as will be shown below, established itself all along the northern littoral of Africa.

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[1] Often this race has been spoken of as the "Armenoid". Cf. Luschan, Ausgrabungen von Sendschirli, Berlin, 1893 and 1911.—Consult the same author's contributions to "Arch. für Anthr.", 1890, "Journ. Anthr. Soc., London", 1911; "Arch. Rass. Ges. Biol.", 1907.—See also Weninger, "Mitt. Geogr. Ges.", Wien, 1920.

If, from among the Semitic people, the Hebrews are to be singled out as especially interesting, I may say that these also are products of the same general racial intermixture, the Near Eastern and the Oriental races being their fundamental stocks. If to-day among the Jews we distinguish two main branches, the Sephardim and the Ashkenazim, ethnological study shows that among the former the Oriental racial admixture predominates, and among the latter the Near Eastern. The differences among the Jews in Europe are to be accounted for by their mingling with the peoples among whom they respectively dwell.[1] The southern Jews, the Sephardim, have been largely crossed with Mediterranean blood, whereas the eastern Jews, the Ashkenazim, have been crossed with Alpine and Mongoloid blood. We may suppose that at the very beginning of the diaspora, the Jews who had been more strongly influenced by a Near Eastern admixture in northern Palestine must have tended to migrate northward, whereas the Jews of southern Palestine, in whom there was a larger infusion of Oriental elements, must have inclined to sail westward across the sea into the countries of southern Europe—so that the two main branches began to become sundered in those very early days.[2] The Sephardim and the Ashkenazim represent different racial mixtures. After what has been said it is almost superfluous to insist once more that we are just as little entitled to speak of a "Jewish race" as of a "Teutonic race", inasmuch as the Jews, no less than the Teutons, are the outcome of particular racial minglings. Nevertheless, we can describe the main racial characters of the Jews and of the Teutons respectively, distinguishing them clearly one from another.

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[1] Cf. Maurice Fishberg, The Jews, a Study of Race and Environment, Contemporary Science Series, Walter Scott, London, 1911.

[2] Luschan, op. cit.—Weissenberg, "Arch. Anthr.", 1909, and "Zeitschr. Ethn.", 1909.—Fishberg, "Amer. Anthrop.", 1902.—Wagenseil, "Zeitschr. Morph. Anthr.", 1922.

3. EGYPT AND NORTH AFRICA

When we turn to consider the anthropology of Egypt, we find that the opportunities for study are exceptionally favourable, seeing that there is an uninterrupted supply of skulls derived from a period lasting more than five thousand years. The earliest prehistoric remains enable us to distinguish two main types in the population of Egypt. One of them is easily recognisable, consisting of an unmistakably Negro population, which used to extend right down into Lower Egypt. Throughout the most flourishing period of the Egyptian realm, the Negroes formed a notable constituent of the Egyptian population, and, as the record of the tombs shows, they were not exclusively of a servile class, but were some of them wealthy and distinguished persons, for Negroids were to be found even in the priesthood and as members of the royal house.

I may mention in passing that from time to time Negro or Nubian rulers sat on the Egyptian throne. This Negroid racial element was intimately mixed with the second constituent of the Egyptian people—the Mediterranean race whose main habitat was and is the south-western parts of Europe. It is probable that Mediterraneans formed the chief ingredient of the population and were consequently the principal sustainers of the Hamitic culture of Egypt. There were, however, manifold additional racial components. A typical "Ramses nose", as it is sometimes called, cannot have been contributed either by the Mediterranean or by the Negroid elements. From ancient Egyptian sources of information, however, we learn of a secular struggle against the desert Semites who invaded the land of the Nile from across the Red Sea. In this way a large admixture of the blood of the Oriental race was introduced into Egyptian veins. We are told, also, of hordes of blond assailants, so from time to time Nordic elements must have been added to the compost. Coloured pictures show unmistakable Nordic types. Finally, skeletal remains include brachycephalic skulls, presumably those of Near Easterns. Realist figurines, some of which represent dwarfed female figures with the pendent breasts and the steatopygia characteristic of Hottentot and Bushman women, show that the Egyptians must have been acquainted with these tribes, and that probably Hottentots and Bushmen must at one time or another have made their way or been brought as far north as the Lower Nile. We may suppose that the racial composition of the Egyptian people must have remained much the same from very early times down to our own day, though the proportions of the various ingredients (especially as regards the Negroid component) may have undergone considerable modifications. The farther up the Nile we go, the more predominant does the Negro type become, and towards the southward limits of Egypt we come to regions whose inhabitants must be described as Negroes with a strong dash of Mediterranean or (as the case may be) as Mediterraneans with a strong dash of Negro.

Westward from Egypt, right across northern Africa, the upper stratum of the population consists to-day of the Islamic stock introduced there by the Arabian conquest. Underlying this, however, and on all hands plainly recognisable (especially in Algeria, Tunisia, and Morocco), are the Mediterraneans, here called "Berbers" but substantially the same as the chief settlers of south-western Europe. Mingled with them are, first of all, Negro elements, and, secondly, Oriental racial elements brought hither by the Arabs. The proportions in the mixture vary to an extraordinary degree from place to place, one racial type being predominant here, another there. This population spread westward from the continent into the Canary Islands lying off the west coast, for the extinct Guanches of these islands, speaking a Hamitic tongue, unquestionably belonged to the Berber stock. But among the Guanches, and probably also in remote valleys of the Atlas Mountains, tall, blond, broad-faced vestiges of the Cro-Magnon race have been preserved.[1] As we move southward from the coastal region of northern Africa towards the desert we find in the population a steadily increasing proportion of Negro blood, which in the end becomes predominant, so that there is a gradual transition to genuine Negro stocks, among which, however, especially in the leading families, we can often discern evidence of a partial Mediterranean ancestry. Just as Hamitic and Semitic philological and cultural elements can be traced through Africa from north to south, so can evidence of the admixture of Mediterranean and Oriental blood with Negro blood be discovered throughout this vast area.

4. AFRICA PROPER

When anthropologists speak of "Africa" without qualification, they generally mean the whole continent with the exception of the northern littoral, that is to say Africa from the Sahara to the Cape of Good Hope, including Madagascar but not the islands lying off the west coast. This area is mainly peopled by the Negroid race. Anthropologically regarded, "Negroland", the whole region inhabited by the Negroid stocks, exhibits the most manifold varieties of culture. Racially these populations have, as already said, been considerably influenced from without by the Mediterranean and Oriental races. Apart from such admixture, the Negroid indigens consist of two clearly distinguishable strata. One of these, the more primitive, comprises the Pygmies, distributed as isolated tribes or groups in various parts of Africa; the Bushmen of the south; the Pygmy tribes in the western Congolese and Cameroon forests; and numerous similar tribes in the Central African primeval forest and in the East African coastal regions. These Pygmies are characterised by their small stature (the average height of the adult males being 1·40 m.), a rounded cranium, a convex upper lip, and (for Negroes) a comparatively fair skin. At one time they ranged over the whole African continent, and even extended into Europe (figurines with steatopygia dating from the European Ice Age). To-day they are restricted to desert regions and to parts of the primeval forest, and the stocks have undergone bodily differentiation.

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[1] Fischer, Bericht der Verh. der anthr. Ges. zu Halle, 1925.

The upper stratum of the Negroid population, comprising what are usually spoken of as "Negroes", are extraordinarily diversified in different parts of Africa, but are none the less to be regarded as forming fundamentally one race, modified here and there by various other racial ingredients. There is no clear anthropological distinction between the stocks to correspond (for instance) with the cultural and linguistic severance into Bantus on the one hand and Nigritics or Sudanese on the other. Stocks belonging to the Sudanese group have for the most part been considerably "Orientalised" or "Mediterraneanised": but we can find Bantu stocks showing similar changes; and, conversely, alike among the Sudanese and among the Bantus there are stocks exhibiting the Negroid type in all its purity. The typical and unmodified Negro has the following characters. The stature is considerable, the average of adult males ranging (according to the stock) from 1·62 to 1·82 m. As regards proportions, we note that the extremities are long, especially the forearm and the leg. The skull is long and narrow, with a peculiarly rounded forehead and occiput, a flattened vertex, and strongly developed jaws. The nose is extraordinarily flattened, wide, with the nostrils showing as almost transverse slits; the cheekbones are prominent; the chin is well developed; the mouth is given a peculiar aspect by the tumidity of the lips. The skin is of a dark-brown tint, varying in its depth; the hair and the eyes are black. The hair on the scalp has a spiral twist; the body is much less hirsute than in most other races; the beard is a comparatively late growth, but when it comes it has the European shape. The mental endowments of Negroids are very different from those of Europeans, being peculiar and characteristic, but I cannot go into details here.

This upper stratum of the Negroids was once, just like the Pygmy stratum, distributed northward as far as the Mediterranean shore of Africa, and beyond it on the southern coast of Europe (the discoveries in the Grimaldi caves). Moreover, there is a good deal in the culture of central Europe during the Stone Age (the stone sculptures of Solutré, for instance) which indicates that the Negroes must have been in close touch with Europe. It was they, obviously, who overran the regions previously occupied by the Pygmies, and restricted these to their present habitats; but we do not know whether they were themselves driven southward by the Mediterraneans, or what precisely in other respects were the relations between them and the Europeans. The human types most closely akin to them anthropologically are to be found in Hindustan and the South Sea Islands, some of the Melanesians of Papua being remarkably like Negroes. The likeness extends to tint of skin, growth of hair, shape of nose, and physiognomy in general; and, to complete the resemblance, we find in New Guinea, as well as the tall Melanesians, Pygmy tribes known as Negritoes. But in the South Seas just as in Africa, the mutual relations between the normal-sized and the diminutive black stocks still await elucidation.

For the sake of completeness we must glance at the Negroes outside Africa, and above all in the United States. In the American Union we find a peculiar intermediate type which has been produced by the indiscriminate crossing of all sorts of Negroid stocks, so that individual characters are chaotically intermingled without the formation of a genuinely new race. The American Negroes have, however, retained the typical Negroid characters, the mental ones not excepted. Where they have been left pretty much to themselves (as, for instance, in Jamaica), isolated groups have relapsed into states of primitive culture (fetishism, voodoo, etc.), and have become what are sometimes termed "Bush Negroes".[1] As far as South America is concerned, it is well known that in various parts of that continent there has been extensive miscegenation between Europeans and Negroes. There the different grades of coloured folk are distinguished as mulattoes, quadroons, octaroons, etc.

5. EASTERN, CENTRAL, AND NORTHERN ASIA

The great mass of the Asian continent, apart from Hindustan, is chiefly occupied by Mongoloids. Anthropologically there is a fairly close kinship between the Eskimos and these; and the indigens of America have likewise affinities, though more remotely, with the Mongoloids. Ethnologists divide the latter into: first, Mongols in the narrow sense, with a northern branch consisting of the Manchus, the northern Chinese, and the Koreans, and a southern branch consisting of the southern Chinese and the Japanese; secondly, the Turco-Tatar stock;

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[1] An area inhabited by Bush Negroes will be found under that name in maps of Surinam, Dutch Guiana. For the remarkable history of the origins of this tribe, see an article in the "Menorah Journal", New York, March, 1928.

and, thirdly, the Ural-Altaics. No trustworthy anthropological classification of these vast populations has as yet been possible, but the above-mentioned groups are distinguishable one from another by a number of anthropological characters, so there can be no question but that the Mongoloids are split up into various sub-races, even if the boundaries between them are not, so far, accurately determinable. Common to all Mongoloids is the Mongolian face, which is flattened but with powerfully developed cheekbones, the flatness of aspect being produced by the way in which the zygomatic processes run forwards parallel to one another until they bend sharply inwards (almost at a right angle) towards one another and into the bodies of the malar bones. The root of the nose is low, and the nose as a whole is broad and flat, though nothing like so broad as it is in Negroids; but we also find Mongoloids with a fairly prominent nose. The eyes are characterised by the previously described (p. 131) Mongolian fold. The skin is tinted by a peculiar yellowish-brown pigment, tending to be light yellow in some of the groups, and verging on brown in others. The Mongolian spot (p. 133) is likewise characteristic of all Mongoloids. The hair is stiff, the individual hairs being very thick and hard, the cross section almost a quadrangle with rounded corners. The stature varies in the different sub-races. The average height of adult males among the northern Chinese is 1·70 m.; among the Tatars, 1·63 m.; among the Japanese, 1·58 m.; and among some of the Lapp tribes, as low as 1·52 m. The shape of the head is also variable, ranging from medium-broad to broad. Among the Japanese, just as among Europeans, we can distinguish two social types; an upper stratum in which the characters are comparatively fine, and a lower in which they are comparatively coarse. In the former the face is narrower and longer, the nose being sometimes small and (for a Japanese) narrow.

The Mongolian stocks have a formidable power of expansion. In prehistoric times, floods of them spread (in a movement which was perhaps connected in some way with the Aryan invasion of Hindustan) over the Malayan Archipelago, to mingle there in the most diversified degrees with the indigenous populations which were themselves already multifarious. The diffusion occurred in two main pulses: the earlier migrants, who were probably less numerous than the later, fused much more thoroughly with the autochthons, giving rise to the Inner or Forest Malays, who have but scanty Mongoloid characters and sometimes none at all; the later migrants helped to produce the Outer or Sea Malays, including the Javanese, the Tenggerese, etc., in whom we can plainly recognise Mongoloid traits conjoined with those of the aboriginal populations.

Another mighty wave, originating perhaps at an earlier date than the former of the two, flowed westward, Mongolising the Slavs, with the results previously described. A very recent outflow is that which has moved eastward across the Pacific into the American Far West, giving rise there to the "yellow peril". The peopling of the Japanese islands was also effected for the most part by a Mongoloid influx from the Asiatic mainland, the indigens of the south being absorbed into the extant Japanese population, whereas those of the north still remain distinct as the Ainu of Yezo and Saghalien. In the Ainu no trace of the Mongol can be discerned, for in their hairiness and in the horizontality of their palpebral fissures they are more akin to Europeans. They may have had a joint origin with the latter from the primal human stock, and have been pushed right across Asia into their present habitat. Some of the racial groups of Hindustan, such as the Todas, may have been similarly derived.

The Eskimos are peculiarly related to the Mongoloids, having narrower skulls and much narrower noses, but being in all their other characters very like Mongols. Presumably they split off early from the Mongoloid stock, and took a special evolutionary trend in the seclusion of their arctic habitat.

The autochthonous population of the Americas may likewise be an offshoot from the same parental stem, seeing that linguistic and ethnographical data combine to show that they have Mongolian affinities. We may suppose them to have crossed Bering Strait from Asia to America subsequent to the Ice Age. Then, gradually making their way southward, they underwent considerable modification after settling down in various localities, thus forming distinct racial groups. The yellowish-brown tint of skin, the Mongoloid proportions, and the Mongolian spot, have been preserved; but the Mongolian palpebral fold has been lost, unless we are to suppose that it had not yet appeared in the joint ancestry at the time when this branch became detached. An especially striking development in the American indigens is the narrowness and length of the nose, which is so marked in some of them as to make that organ strongly aquiline.

6. AUSTRALIA AND THE PACIFIC

Very little can be said here about the South Sea Islands and Australasia, but there must be some brief mention of these regions, were it only to show the extreme plasticity of the human species and how large is the number of the varieties into which it has differentiated. Here and there in Hindustan, in Ceylon, and in some of the islands of the Malay Archipelago, we still find vestiges of the racial stratum of the Veddahs, an extremely primitive stock. They are small (though not so small as the Pygmies), with broad faces, squat noses, and wavy hair resembling that of Europeans. The Australian Blackfellows are of a somewhat similar type, but clearly distinguished by the shape of the skull, a much more considerable stature, and a different shape of beard. The skulls of these Australian aborigines are more primitive in structure than those of any other extant breed of man. Another stock resembling the Veddahs is that of the Negritoes, found in the Philippines, New Caledonia, etc., who, however, contrast markedly with the Veddahs in having frizzy instead of wavy hair. They are very small, the average height of adult males among the Philippine Negritoes being 1–42 m. The Melanesians of Papua (and the Tasmanians, who became extinct during the nineteenth century) are like the Negritoes in many respects, including the frizziness of the hair, but are much taller, and have a different shape of skull.

The South Sea islanders in general, classed promiscuously as Polynesians, are of a very different type, having straight hair and a much fairer skin. In many respects, such as their bodily proportions, their physiognomy, and the growth of their hair, they resemble Europeans.

Springing from the common human root, all these stocks have to some extent remained primitive, and have to a greater or lesser extent undergone peculiar developments of their own. The South Sea tribes have intercrossed in numberless ways.

7. CONCLUSION

In this work it has only been possible to give a very brief sketch of the racial types—such a sketch as was essential in amplification of the foregoing section. I have shown, however, that *in human heredity the innumerable characters which differentiate individuals and groups (races) are tenaciously and inalterably transmitted from generation to generation*; and my aim has been to point out the manner in which such inheritable peculiarities coalesce in specific ways to mark off the various "races" of man each from the others. *Technically speaking, there is no such generalised being as "man"; there are only men and women belonging to particular races or particular racial crossings.*

SECTION THREE

MORBIFIC HEREDITY FACTORS

by

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CHAPTER EIGHT

THE CONCEPTS HEALTH, DISEASE, AND NORMALITY

It is characteristic of living beings that alike in their structure and in their modes of reaction they should in general be adapted to their customary environment; and we say that an organism is adapted to its environment when its structure and the vital manifestations dependent on that structure ensure the maintenance of its life in the said environment.

Not infrequently, however, we encounter living organisms which (it may be as an outcome of external influences and it may be on account of faulty structure) fail, more or less, to exhibit the desirable adaptation, so that their chances of maintaining their existence are impaired. A creature which is living on the frontier of its adaptability is said to be diseased. Thus there are all possible gradations between perfect health and severe illness. There is no fundamental biological distinction between health and disease.

Perfect health is a condition of perfect adaptation, and an organism is diseased in proportion to the extent to which its adaptability is impaired. When from internal or external causes it is forced over the borderline of its adaptability, death ensues. The dead body no longer manifests adaptive reactions, and is thereby distinguished from the living body. *By disease*, therefore, *we mean the state of an organism which is on the borderline of its capacity for adaptation.*

Slight deviations from the condition of full adaptability are not yet termed illness or disease. A defect in colour vision, for instance, is not an illness but an anomaly. By anomalies we mean permanent deviations from a state of perfect adaptation which, though they do in some measure restrict the capacity for self-preservation, are not serious enough to entail a direct menace to life, being still a good long way from the borderline of the capacity for adaptation. Many anomalies, however, are also predispositions to disease—as, for instance, are the so-called diatheses (susceptibilities) which will have to be discussed in due course. Organisms thus affected are abnormally prone to be approximated to the frontier of their capacity for self-preservation, and thus to be made ill.[1]

For a long time it was usual to imagine that every kind of living organism is endowed with a definite "normal range of variation", and that deviations from the mean type must be regarded as normal so long as they keep within that range. On the other hand, deviations transcending the "normal range of variation" were to be regarded as morbid. But in actual fact the "normal range of variation" cannot be defined, for when we are asked to define it we only argue in a circle if we say that it is transcended wherever disease begins.

It is also a grave error to consider without further ado that the average type of a population is the standard of the normal. The average may be far from having the greatest capacity for adaptation. For like reasons, we must refrain from taking the commonest type as the standard of the normal. In a population most of whose members have a goitre, we shall still be disinclined to speak of wens as normal. We must, therefore, dissent from Rautmann's proposal[2] to regard as normal all specimens of most frequent occurrence and up to the mean of the deviation in either direction. No less unsatisfactory is the definition given by J. Bauer[3]: "Normal means average; normal is that development of a character or quality which corresponds or is adjacent to the mean value in the curve of variation." The logical inference from such a definition would be that all populations must contain the same proportion of normal individuals, and that no one of them could be looked upon as more anomalous or more degenerate than any other. Moreover, a whole population of normal individuals would no longer be a theoretical possibility, whereas the definition of the normal should certainly leave such a possibility open. If in any population all the abnormals were to be eliminated, then, on Bauer's definition, some of those previously accounted normal would have become abnormal; and, conversely, those hitherto deemed abnormal would become normal if the normal were to be eliminated. This train of reasoning should suffice to show the absurdity of a view which has of late years gained undeserved currency. Thus we must agree on the whole with Hildebrandt's criticism of the attempt to make the ideas of normal and average coincident,[1] although we cannot follow him all the way in his own attempt to define the concept of normality. We must consider erroneous all attempts "to provide unified marginal values for the normal" (Rautmann). It is a prejudice to suppose that a distinct normal type or "normotype" must exist. The problem of the delimitation of the normal is not one of ascertaining a content, but simply and solely one of definition. Those only who fail to understand this, will try to discover the "essence" of normality. Definitions must be freely (though not lawlessly) drafted. One who formulates them must have a clear vision of their consequences, and he must pay due heed to linguistic usage. My own definition, which assumes vital efficiency as the conceptual standard, complies with both these requirements. In my view it is by no means necessary that a population should contain only one normal type. Several types, all clearly distinguishable one from another, may be exceptionally fitted to survive; and when we are considering the survival of a people as a whole, we must not leave the division of labour out of account. For the survival of a folk-aggregate it is no less essential that it should contain persons fitted to assume the intellectual leadership than that it should contain hewers of wood and drawers of water, and a community which included persons only of the one kind or of the other would be less efficient and therefore less likely to survive than a community which included both.

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[1] M. von Pfaundler defines these concepts in much the same way as myself. See his Was nennen wir Konstitution, Konstitutionsanomalie und Konstitutionskrankheit? "Klinische Wochenschrift", 1922.

[2] H. Rautmann, Untersuchungen über die Norm, Fischer, Jena, 1921.

[3] Vorlesungen über allgemeine Konstitutions- und Vererbungslehre, Springer, Berlin, 1923.

Mediocrity must not be uplifted to the rank of normality. Even if this were to be accounted permissible in the bodily domain, in the mental sphere its results would be disastrous.

It must however be admitted that there are certain relations between normality and averageness. The most frequently extant average types will in general be well fitted to survive—for they are frequent precisely because they have shown themselves possessed of the power to survive. On the other hand, marked deviations from the average will usually be morbid; but this cannot be taken as a universal rule, and for that reason it cannot guide us in our definition of the normal. The numerous attempts to discover the characteristics of normality (attempts which have of late years produced a voluminous literature) remind us of earlier endeavours to discover the "signs of degeneration". It is certainly possible to note bodily measurements, proportions, etc., which enable us with considerable justification to infer that the bodily structure is normal, and they may therefore be classified as "signs of normality". But such signs are not wholly to be trusted. Persons whose measurements conform to the average may be diseased, and persons exhibiting marked deviations from the average may be normal.

[1] K. Hildebrandt, Norm und Entartung des Menschen, Sibyllenverlag, Dresden, 1920.

Nevertheless, even if we reject the idea of a Procrustean norm as a general standard for all mankind, we cannot follow Grote when he writes: "The individual is the measure of his own normality."[1] Yet we cannot say that such a proposition is "fallacious", seeing that here, as already said, the elements for a precise statement are lacking.

It seems to me that in the last resort the definition of the concepts disease and health must be made dependent upon racial rather than upon individual survival. Sterility, for example, is almost universally regarded as morbid, although it does not in any way impair the individual's powers of self-preservation. On the other hand, pregnancy, childbirth, and lying-in, bring in their train unavoidable risks to the mother's life, and the parturient woman has to go to bed just as if she were ill, but no one includes pregnancy and childbirth among diseases. These processes are essential to the preservation of the species, and we therefore account them normal. Nor are infancy and old age considered to be anomalies, although at these periods there is less capacity for adaptation than during the middle periods of life. The individual's death in old age is normal, for it does not interfere with racial preservation; and the renewal of individual lives by birth is essential to race-survival. Thus every adaptation upon which we make the concepts of health and disease turn, is in the last analysis directed towards the preservation not of the individual but of the race. Individual-survival is only a means to the end of race-survival. Even those mental endowments which impel the individual to self-sacrifice (as in war time) are not morbid, but normal in the highest sense of the term, insofar as they contribute to the preservation of the race.

Of course there is no such thing as absolute or unconditional adaptation. Organisms are only adapted to life in a particular environment. A Negro born in tropical Africa with the northern European's low-grade capacity for resisting heat would be morbid, not normal, and a northern European with a Negro's sensibility to cold would likewise be abnormal. We therefore take as our standard of the normal the adaptability to what happens to be the environment of the race, and it follows that such a standard must differ from race to race.

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[1] L. R. Grote, Grundlagen ärztlicher Betrachtung, Springer, Berlin, 1921.

My definition of disease does not pretend to give a new content to the idea. I do not agree with those pathologists who say that the aim of pathology is "to discover the essential nature of disease". When we look the matter clearly in the face we see that the problem as to the "essential nature of disease" is really nothing more than a problem of definition or terminology; and definitions are not in themselves either true or false, but only more or less fitted for their purpose. The definition I propose harmonises with current linguistic usage, insofar as such harmony is practicable where scientific definitions are concerned. Even the layman, when confronted with a case of illness, is chiefly interested to know whether the patient will live or die! My definition, however, is equally accordant with the technical usage of medical science. Virchow was already inclined from time to time to lay stress on "the character of the danger" entailed by a disease.[1] My definition, therefore, does not involve any fundamental innovation, but only what I regard as a more effective formulation—one which throws a plainer light on the way in which the concept of disease is rooted in general biology. I certainly cannot agree with those pathologists who declare that morbid processes and morbid states are so radically different that we are not really entitled to apply the same word "morbid" to both. In this respect I follow Klebs, who as long ago as 1887 wrote in his *Allgemeine Pathologie*: "These are two series of phenomena between which there are very important differences, and yet they are both equally entitled to the name of morbid or pathological—morbid processes and morbid conditions."

This way of regarding the concept of disease serves also to liquidate the objection that there can be no such thing as a hereditary disease because a disease is always a process and never a state and because processes cannot be hereditary. If it be held improper to speak of "hereditary diseases", then we must withhold the name of "disease" from diabetes and many other ailments which are usually spoken of as "diseases", not only in ordinary life, but also in the terminology of medical science. Surely it is better to formulate the

[1] A. Jarotzky, in the "Münchener Medizinische Wochenschrift", no. 39, 1924, claims priority for making the concept of disease turn upon the notion of capacity for adaptation. It would seem, however, that the Russian clinicist A. A. Ostrumoff was really the first, in the year 1895, to define disease as a state of insufficient power of adaptation. But so far as I understand Ostrumoff and Jarotzky, these authorities do not mean by adaptability precisely what I mean when I use the term. I am thinking of the adaptability which favours the chances of survival.

concept of disease in such a way that it will include these troubles? Fully developed morbid processes (of which inflammation of the lungs may be regarded as a typical instance) will likewise be covered by our definition of disease. For if a man is suffering from inflammation of the lungs he is in respect of his then condition one of the weakly variations, and is accordingly in imminent danger of death.

CHAPTER NINE

SIGNIFICANCE OF MORBIFIC HEREDITY FACTORS IN THE CAUSATION OF DISEASES OF VARIOUS ORGANS

1. GENERAL CONSIDERATIONS

As a rule the causes of an illness are composite. They do not consist exclusively of either environmental influences or of hereditary predispositions; it is usually found that factors belonging to both classes have been at work. Very seldom, however, do we find in such cases that the factors of the two categories have been equally important, seeing that generally those of one kind or those of the other will have been mainly determinative. Thus in most of the diseases termed infectious or contagious the decisive cause is the invasion of the body by certain minute organisms which multiply there to its detriment. On the other hand, *when we speak of hereditary diseases we mean those in whose production morbific heredity factors have been chiefly operative.*

A noted clinician, W. Clausen, who is also famous for his researches on heredity, has recently declared that when we say that a certain illness is the result of hereditary predisposition we have by no means discovered its true cause, since we are not thereby informed regarding the seat and the substratum of the disease. I look upon this objection as far-fetched. The "seat" and the "substratum" of a disease are matters which concern its anatomical aspect; whereas the cause of a hereditary disease is really something that arises out of the nature of the hereditary equipment (if we are looking on the disease as a manifestation peculiar to the individual suffering from it), or something that depends upon a morbid mutation or idiokinesis (if we are looking on it as a manifestation affecting the whole kinship). I consider, therefore, that the pathology of heredity is the first and most fundamental portion of etiological pathology, and that it ranks equally as a topic of research beside the study of the environmental causes of disease.

In the nomenclature of diseases there has been a tendency to imitate the binary nomenclature introduced into biology by the Swedish botanist Linnaeus. Thus when people speak of typhus abdominalis (abdominal typhus, in England usually called "typhoid" or "enteric fever") and typhus exanthematicus (spotted typhus, in England now usually called "typhus" without qualification, though in former days typhoid and typhus were confounded under the latter name) they are apt to think of typhus as if it were a generic and of abdominalis or exanthematicus as if it were a specific appellation. Similarly with "asthenia universalis" and other terms of the kind. The biological affinities are, however, spurious, for we cannot divide maladies into genera and species. The biological classification of diseases caused by microscopic organisms must, rather, be based upon that of these organisms. Typhus abdominalis, being brought about by a vegetable organism, the typhoid bacillus, has no close biological affinity to typhus exanthematicus, in which the exciting cause is a protozoon. Where, on the other hand, we have to do with hereditary disorders, we are concerned with varieties of the human species, so that biologically we can speak of Homo sapiens var. asthenicus.

Morbid hereditary equipments are transmitted in accordance with the same laws as normal ones. This must, indeed, be self-evident to any one who has realised that no essential biological distinction can be drawn between disease and health. Since many morbid heritages distinguish very plainly those who are affected by them from other members of the population, it has been easy to trace their inheritance. It is, in fact, by the study of morbific heredity factors that we have been most satisfactorily enabled to prove that the mendelian laws of inheritance are valid for human beings.

The experimental study of certain organisms, and especially of the fruit-fly (Drosophila) and the snapdragon (Antirrhinum), has shown: on the one hand, that the great majority of the mutations as yet known are more or less morbid; and, on the other, that morbid mutations can in almost all instances be classified as singly dominant or recessive in inheritance. We may naturally anticipate that this will apply also to morbific heredity factors in man, and experience confirms the supposition. Conversely, experimental research as concerns the crossing of naturally extant (non-morbid) races and species shows that the differences between them are not usually determined by single heredity factors (monomerically) but by a number of such factors (polymerically). In the case of man, likewise, the study of the crossing of normal human races and that of the inheritance of normal human endowments, confirms this view. We can, therefore, formulate the rule: morbid hereditary conditions are usually induced by single heredity factors (monomerically), and normal qualities by many such factors (polymerically). This is easy to understand in view of the following considerations. If a particular heredity factor brings about so marked a deviation from the average that the person thus affected becomes plainly distinguishable from other individuals (and such is the essential nature of monomeric characters), the deviation will generally involve an impairment of the capacity for self-preservation, this meaning that it will induce a morbid state. But heredity factors which are not potent enough acting singly to give rise to a distinctive character, and can do no more than bring about a moderate change in some feature, so that only when a number of such factors cooperate is a well-marked deviation produced—will usually have no pathological significance, and will only cause differences that remain within the confines of what is ordinarily spoken of as the normal.

Misled by the obvious workings of heredity where morbific heredity factors are concerned, people have sometimes been inclined to talk of the hereditary transmission of disease as a peculiar fatality. There is no warrant for any such view. The inheritance of normal and of morbid endowments is equally determined. We must never forget that the foundations of every living organism are laid in these hereditary endowments, and that environmental influences can do nothing more than collaborate in their development.

In the present chapter we shall consider only those hereditarily transmissible diseases and anomalies which are of great practical or theoretical importance. No attempt will be made to achieve completeness in the way of describing all the rare conditions which have been shown to be inheritable. Inasmuch as this book is intended for cultured general readers as well as for medical practitioners, is intended for all those who are keenly interested in the health and the future of our race, I have done my best to describe the diseases mentioned in terms comprehensible to laymen. Rarities whose interest is chiefly medical, and whose description necessitates the use of technical terminology, are confined to the paragraphs in smaller type. It would have been misleading to strain towards completeness by giving exhaustive lists of hereditary diseases and anomalies, for in such lists serious and trifling, frequent and infrequent, conditions are lumped together indiscriminately and given ostensibly equal values. Besides, the non-expert reader will be apt to infer that heredity plays no part in the causation of diseases which do not happen to be mentioned in such enumerations.

The study of hereditary diseases has made enormous advances of late years. Many of the problems which could only be mooted in the second edition of this book, published no more than three years before the present one, have been solved in the interim. It has therefore been necessary to revise, and in many instances to enlarge, all the subsections of this chapter. I do not think anything like the same amount of revision will be needed in later editions, for a certain degree of stabilisation is now becoming manifest in our science. Since the literature of the topic is so abundant, and new contributions to it are so easily overlooked, I venture to ask the writers of monographs who would like them to receive due attention when a new edition is called for to send them to me at Herrsching near Munich.

2. HEREDITARY DISEASES OF THE EYE

A description of hereditarily determined maladies and anomalies may appropriately begin with an account of hereditary affections of the eye. We know more about morbid hereditary endowments of the eye than about those of any other organ, and for good reason. The eye is the most complicated and the most important of our sense organs, so that comparatively slight anatomical disorders of the structure of the eye give rise to disproportionally extensive disturbances of function. Furthermore, the eye is exceptionally open to inspection, and can be especially well subjected to medical examination.

We are indebted to ophthalmologists for a number of valuable and comprehensive monographs on the hereditary affections of the eye.[1] As the publications relating to this topic are widely interspersed throughout medical and surgical literature, none but specialists are likely to be able to secure an exhaustive view of them. My own account of hereditary diseases of the eye is, therefore, substantially based on the work of Clausen, who gives an elaborate bibliography. I should also refer to the reprint of all hitherto published genealogical trees showing the hereditary transmission of disorders of the eye which we owe to the labours of the Galton Institute in the University of London.[2]

The heredity of the tint of the eye (the colour of the iris) has—morbidity apart—already been considered in the foregoing section by Fischer. The complete lack of pigment in the eye is, however, a morbid manifestation, whether it appears as a feature of general albinism or as restricted to the eye. Inasmuch as the morbid disturbances dependent upon general albinism centre in the eye, *albinism* can suitably be discussed among maladies of the eye.

In this condition, from youth upwards, the hair is snow-white or yellowish-white from lack of pigment. The skin is also void of pigment, and therefore has a rosy-white tint because the blood circulating in its deeper layers shows through the semi-transparent superficial ones. In like manner the iris, being transparent owing to the absence of pigment, is made reddish in appearance by the bloodvessels which are seen through it; and the pupil, which ought to be black, is also made reddish by the lack of pigment in the retina. The pigment in the eye serves normally to protect the sensitive retina from excess of light, and for want of this protection albinos are unduly sensitive to light—are "photophobic". In Germany, albinos are often nicknamed "Kakerlaken" (cockroaches) because, like these insects, they tend to scurry away from a bright light. They usually go about with their heads hanging, their brows puckered, and their eyelids half closed.

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[1] Special mention may be made of W. Clausen's Vererbungslehre und Augenheilkunde, "Zentralblatt für die gesamte Ophthalmologie", vol. XI, 1925, no. 3.

[2] Treasury of Human Inheritance, vol. II, Heredity of Eye Anomalies and Diseases, Cambridge University Press, London, 1922. (Only the first part has as yet been issued.)

The photophobia of albinos is apt to be accompanied by rhythmical twitchings of the globe—nystagmus. The morbidity of albinos is in large measure dependent upon the way in which they are dazzled by anything stronger than a half light.

The heredity of albinism has been carefully studied by Karl Pearson, E. Nettleship, and C. H. Usher,[1] and also by C. Seyfarth.[2] In all, nearly seven hundred genealogical trees of families in which albinism occurs have been published. A study of these shows clearly that albinism is always simply recessive in its heredity. I reproduce here a genealogical tree published by Tertsch,[3] which is also instructive as regards the heredity of recessive characters in general.

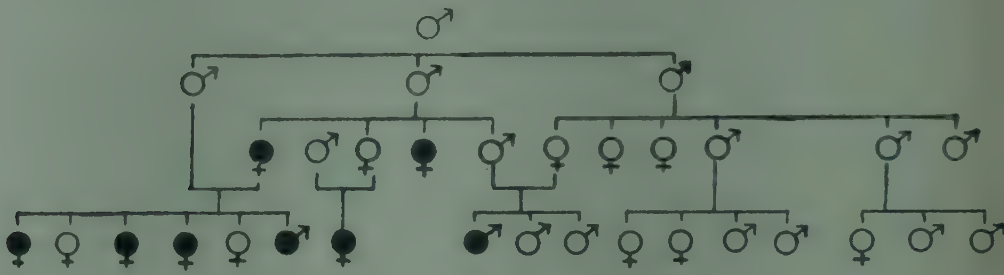

FIG. 32.
General albinism, after Tertsch.

In the foregoing tree we see to the left a marriage between uncle and niece, producing six children, four albinos and two normals. The albinotic mother is to be regarded as homozygotic, the father as heterozygotic. Through the marriage of these two persons of near kin the recessive taint has been contributed by both parties. In the middle of the same tree we see a marriage between first cousins. Of their three children, one is an albino and two are normal. Since both the parents are healthy, we may presume them both to be heterozygotic. The morbid character can certainly not be dominant, for in three marriages albinotic children are born from parents both of whom are healthy. General albinism is, rather, to be deemed recessive, and appears to be due to the absence of a heredity unit which in the account of mendelian heredity in rabbits was on p. 58 denoted by *A*. The tree also shows very plainly the importance of the marriage of near kin in making recessive taints manifest. The statistics indicate that about a third of all albinos are the offspring of consanguineous marriages, whereas the general frequency of such marriages does not amount to one per cent. Exceedingly instructive are cases in which albinos are born of incestuous unions. Two such instances are shown in Fig. 33 and Fig. 34 respectively. Of course the danger that there will be a coalescence of like taints in the two parents is especially great when they are connected by blood within the "prohibited degrees".

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[1] Albinism in Man, 3 vols, Dulau, London, 1911.

[2] Beiträge zum totalen Albinismus, "Virchows Archiv", vol. CCXXVIII, 1920.

[3] Albino mit bemerkenswertem Stammbaum, "Zeitschr. für Augenheilkunde", vol. XXV, 1910.

In addition to albinism extending to the eyes as a part of general albinism, albinism restricted to the eyes may occur. In such cases the pigmentation of the skin and the hair is normal, but the morbid manifestations in the eye are identical with those just described. A very remarkable feature of these localised instances of albinism is that they are met with only in males. But women, though themselves outwardly unaffected, can transmit the taint from their fathers to their sons, and do so (as the mendelian theory would lead us to expect) to half of these on the average. Their daughters, on the other hand, show no sign of the affection, but it may be transmitted in the female line in a latent form for two, three, or more generations, to crop up at length in male issue. Men never transmit it to their sons, but can do so indirectly to male grandsons by way of their daughters. Thus the course of inheritance is said to be recessively sex-linked. As is usual in recessive heredity, so here, the parents of affected individuals are as a rule ostensibly

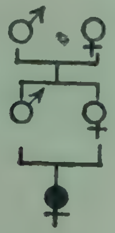

FIG. 33.
Albinism, after Bemiss.[1]

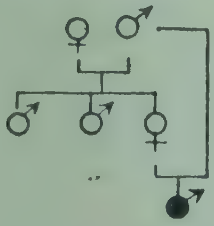

FIG. 34.
Albinism, after Pearson.

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[1] S. M. Bemiss, Report on Influence of Marriages of Consanguinity upon Offspring. Transactions of the American Medical Association, vol. XI, Philadelphia, 1858.

normal. The peculiar sex-linkage can be explained on the assumption that the morbid manifestation is due to the lack of a gene[1] which is normally contained in the sex chromosome (see above, pp. 82 et seq.). Since a man has only one sex chromosome, a defect which is sex-linked in this way will manifest itself in him without more ado; but in the female, who has two sex chromosomes, the defect of one sex chromosome will be marked by the corresponding normal gene in the other sex chromosome. The following genealogical tree will help the reader to picture the course of events:

This tree shows us how the trouble from which the grandfather suffers recurs in several male grandchildren, and how it is transmitted to a great-grandson through two generations of outwardly unaffected females. The defect is not dominant, seeing that its hereditary transmission cannot be traced in an unbroken line, as happens in the case of dominant disorders.

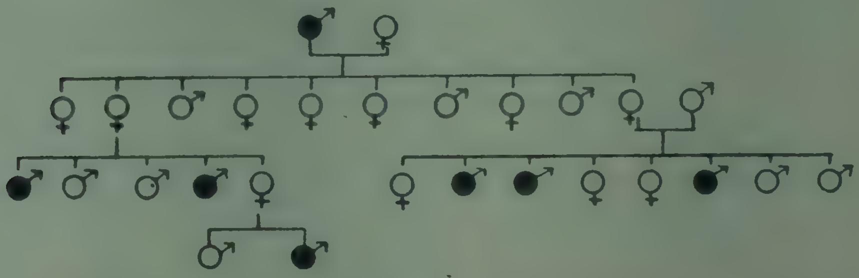

FIG. 35.

Albinism of the eye with nystagmus, after Mansfield (abbreviated, some of the females of the second generation having been omitted).

Jablonski[2] has described a family in which 27 persons belonging to 3 generations suffered from slight albinism (albinoidism). Most of the persons thus affected became darker as time went on. We may suppose them to have been heterozygotic, and that the homozygotic coincidence of two such taints would cause a much severer albinism (provided that such individuals were viable). But in heterozygotics the taint would only show itself with moderate intensity, and in the course of years a change towards dominance would occur. Such a disposition, able to manifest itself even in heterozygotics, must be regarded as more strongly morbific than a completely recessive albinotic taint. An account of three other families affected with albinoidism has been published by H. Zimmermann.[1]

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[1] See footnote to pp. 73–4.

[2] Ueber Albinismus des Auges, "Deutsche Medizinische Wochenschrift", no. 26, 1920.

Associated with the recessive nature of the heredity is the fact that general albinism tends to show itself with especial frequency in certain localities where marked inbreeding occurs. Thus in the island of Veglia on the Dalmatian coast, which has a population of about 20,000, Hanhart found a number of albinos, though the usual proportion is no more than 1 : 10,000 or 1 : 20,000.

Partial albinism, in which the skin is flecked with white here and there, will be considered under hereditary diseases of the skin.

Among pigmentary anomalies of the eye, mention should be made of what is termed *heterochromia,* in which one eye is brown and the other bluish-grey. This condition has some pathological significance, for the lighter-coloured eye in such persons is prone to a peculiar creeping inflammation. Gossage reports an instance, recorded by Dr. Walter Bell, in which heterochromia iridis could be followed in one family for five generations.[2] This observation has an important bearing on the theory of heredity, for it shows that a one-sided character can undergo typical inheritance. We have to thank G. J. Bond for a detailed account of heterochromia in man and other animals.[3] It has been possible to breed heterochromia in cats, dogs, and guineapigs. In many instances heterochromia seems to be a particular manifestation of hereditary partial albinism, so that it shares the irregular heredity of that affection. In human beings and domesticated animals we not infrequently see brown spots in an iris whose basis tint is grey or bluish-grey. This phenomenon probably belongs to the domain of birthmark-formation, the heredity of which will be subsequently discussed.

As a local manifestation of a hereditary constitutional anomaly whose pathological significance depends upon undue fragility of the bones (fragilitas ossium) we encounter a remarkable bluish-grey tinting of the sclerotic (the membrane which makes up what in popular parlance is termed the white of the eye). The bluish-grey tint is due to an abnormal thinness of the corium. The constitutional anomaly which underlies it will come up for consideration later, since the brittleness of the bones is the leading morbid characteristic of the whole affection.

It has long been known that *errors of refraction* in the eye are to a great extent hereditarily determined. Refraction depends upon the cooperation of various parts of the eye: upon the length of the main axis of the globe; upon the curvature of the cornea; upon the curvature of the lens; and so on. When the eye has to be accommodated for near vision, the lens must be more strongly curved, for otherwise the image of the object concerned would fall behind the retina, and vision would consequently be indistinct. This accommodation is effected by a special muscular effort. But the eyes of a considerable proportion of human beings are when in repose (without the muscular effort of accommodation) already adapted for near vision, so that the images of more distant objects are formed in front of the retina, and distant vision is indistinct even when the accommodation is relaxed. Such persons are said to be *short-sighted* or *myopic*. There are various degrees of short-sight, and the minor degrees do not cause very much disturbance of vision—all the more seeing that the refraction can be fully corrected by suitable spectacles.

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[1] Ueber menschlichen Albinismus, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XV, no. 2, 1923.

[2] A. M. Gossage, The Inheritance of Certain Human Abnormalities, "Quarterly Journal of Medicine", April, 1908, p. 341.

[3] On Heterochromia Iridis in Man and Animals, "Journal of Genetics", vol. II, 1912.

Till recently it was generally believed that short-sight was caused by the excessive and prolonged use of the eyes for near vision. Because a marked increase of short-sight was often noticed during the school age, it became customary to speak of "scholastic myopia". This theory has of late been exploded, above all by the comprehensive researches and able reasoning of the Zurich ophthalmologist A. Steiger.[1] It may now be regarded as proved that the increase of short-sight so often observed in youth is mainly the outcome of internal causes. The statistical evidence which has been brought forward to show that school work, or other prolonged use of the eyes for near vision, causes short-sight, will not bear critical examination; and it is time to abandon the idea that myopia is a sort of petrified adaptation of the eyes to close work. Above all it is erroneous to attribute to what we may presume to be an individually acquired adaptation the origination of the hereditary tendency to short-sight. Such notions are quite incompatible with the results of the study of heredity.

Most ophthalmologists and school hygienists are to-day inclined to take an intermediate position upon this matter, accepting the view that the tendency to short-sight is hereditary, but considering that it can be roused into activity by near work. This obvious and seemingly cautious hypothesis is hard to refute, but it cannot be regarded as established upon a sure foundation. This much, at any rate, would appear to be certain: 1. short-sight never arises in the absence of hereditary predisposition; 2. when predisposition exists, short-sight (extreme or moderate) may arise in persons who are not engaged in near work; and, 3. it remains questionable, to say the least of the matter, whether near work can promote the activation of an extant tendency to the development of short-sight.

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[1] Die Entstehung der sphärischen Refraktionen, Berlin, 1913.

The inheritance of short-sight does not run the same course in all families. To put the statement in other words, the clinical picture of short-sight does not coincide with any one biological scheme, but is coextensive with several such schemes. In Halle, during recent years, Clausen has collected hundreds of genealogical trees showing the incidence of myopia, and has thereby been led to infer that short-sight is recessive. One of these trees is reproduced in Fig. 36. A recessive heredity for this anomaly is indicated by the circumstance that short-sighted offspring are often born of normal-sighted parents, and by the circumstance that when both parents are short-sighted their children invariably suffer from the same defect. W. Jablonski also holds the opinion that the heredity of short-sight is recessive.[1] Making use of Weinberg's "proband method" (see below, pp. 507 et seq.), he found among the brothers and sisters of 31 persons affected with extreme short-sight $16 \cdot 5 \pm 3 \cdot 14$ % of short-sighted individuals, when both parents

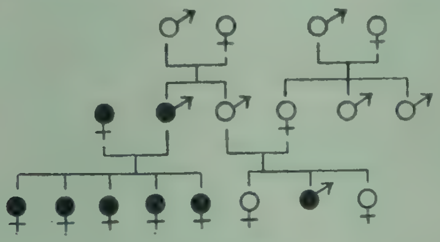

FIG. 36.
Short-sight, after Clausen.

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[1] Zur Vererbung der Myopie, "Klinische Monatsblätter für Augenheilkunde", vol. LXVIII, 1922.

were normal-sighted. But among the brothers and sisters of 12 persons affected with extreme short-sight one of whose parents was also extremely short-sighted, there were 40·8 ± 6·2 % of short-sighted individuals. These results harmonise with the theory that the heredity of short-sight is recessive, for the theoretically calculated percentage should be 25 in the former case and 50 in the latter. The margin of error does not exceed what might be expected in view of the small number of instances dealt with in this enquiry (see below, p. 508). On the other hand Vogt, the Zurich ophthalmologist, points out that extreme short-sight is so common in the children of short-sighted persons that the assumption of a recessive heredity will not wholly account for the facts.[1] In Fig. 37 I reproduce from Jablonski a genealogical tree in which the incidence of short-sight can be traced back for three generations in both parental stocks of three short-sighted persons. Now it is certainly correct to say that such a series of cases might present itself even if the heredity were recessive, assuming that in the ostensibly normal parent the tendency to short-sight also existed, though masked. Short-sight is so common an anomaly that it might very well appear for several generations in succession. If we assume that 10 % of all human beings are affected with a high degree of short-sight, then, if the heredity of short-sight were simply recessive, more than 50 % of those whose sight is normal must have a hereditary tendency to

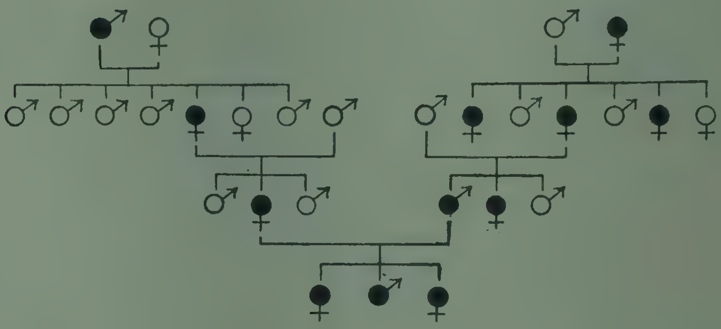

FIG. 37.
Short-sight, after Jablonski.

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[1] A. Vogt, Ueber Vererbung von Augenleiden, "Schweizerische Medizinische Wochenschrift", nos. 7 and 8, 1923.

short-sight though in a masked form.[1] But the frequency with which manifest short-sight can be traced through several generations seems to me to indicate that there must be dominant as well as recessive heredity factors of this anomaly. In 95 cases of short-sight where the family history was well known, Steiger was able in 70 instances to find that short-sight had existed in one or both parents also. Many of the genealogical trees showing the family incidence of short-sight are of such a character as to suggest that the heredity was in part determined by the influence of what are called irregularly dominant factors—this meaning, factors which usually manifest their effects quite simply, but whose working is sometimes inhibited by the operation of other heredity factors or by that of environmental conditions. Of course if this be so, then the appearance of short-sight may sometimes be due to the cooperation of dominant and recessive heredity factors.

Fleischer studied the inheritance of short-sight in a Wurtemberg village. In seventeen families where one of the parents suffered from high-grade myopia, about half of the children were short-sighted. The observation affords a certain amount of evidence to show that in these cases the short-sight was determined by a single gene, that is to say that the tendency was a dominant character. Moreover the genealogical tree which Clausen, in the previously mentioned work, has constructed out of Fleischer's data corresponds most nearly to the conception of an irregularly dominant heredity. In like manner the genealogical tree shown in Fig. 38 speaks in favour of dominance. We must admit, however, in view of the frequency of a tendency to short-sight, that these genealogical trees are also accordant with the idea of a recessive heredity as understood by Clausen and Jablonski. The problem of the hereditary determination of short-sight will presumably have a fuller light thrown on it ere long by the elaboration and publication of the numerous genealogical trees which Clausen has collected. The Berlin ophthalmologist Czellitzer has also collected many (more than a thousand) genealogical trees showing the incidence of extreme short-sight, but he has not as yet published even a selection of them, having a bias in favour of the study of heredity by the recording of massed statistics obtained by the "brother-and-sister method".[2]

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[1] For details as to these numerical relations, see p. 510 in the chapter on Methods.

[2] Explained below on pp. 507 et seq.

Anatomically, short-sight may be induced by two different anomalies, undue length of the globe and excessive curvature of the cornea. Steiger's investigations prove that other alleged causes are devoid of practical moment. Now, Jablonski has shown that an abnormal length of the eye is a recessive character as against abnormal shortness. This would explain why myopia dependent upon excessive length of the globe is usually recessive. Myopia resulting from undue curvature of the cornea might behave in a different way genetically. Jablonski, acting on a suggestion made by the geneticist Poll, has endeavoured to discover the range of modification in normal refraction, studying for this purpose the refraction in 52 pairs of twins. (This method of studying genetics by the examination of twins will be further considered in the chapter on Method—see below, p. 554.) He found that mon-oval twins rarely differ from one another in their refraction by more than two diopters. (The diopter is the unit ordinarily employed in the measurement of refraction. It represents the refractive power of a lens having a focal length of one metre.) Although it is impossible to ascertain in this way the maximal range of modification, we are entitled to say that short-sight of more than two diopters must almost certainly be hereditarily determined. Of course this must not be taken as implying that myopia of a lesser grade, ranging say from one half to one diopter, may not likewise be hereditarily determined. When in one of Clausen's genealogical trees we find in two members of the same family and belonging to the same generation that one has short-sight amounting to 8 diopters and another short-sight amounting to only 1·5 diopters, it seems hard to suppose that the two anomalies are to be regarded as genetically identical. But there is nothing to prevent our assuming that a particular heredity factor may heterozygotically determine a moderate degree of short-sight (and produce a picture of "dominant" heredity), while homozygotically determining a high degree of short-sight (the heredity being here "recessive").

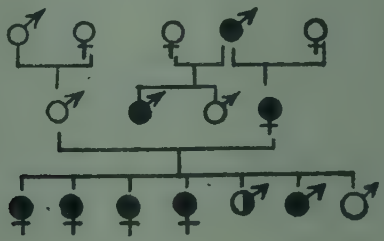

Fig. 38.
High-grade myopia, after Czellitzer.

Moreover, in cases of high-grade myopia the range of modification might be more than 2 diopters, so that in a brother-and-sister series recorded by Fleischer the respective myopias of 7 diopters and 15 diopters might in the circumstances be genetically homologous.

Some genealogical trees have been published showing recessive sex-linked heredity (by Worth and Oswald, for instance, the literature being given by Clausen); but such heredity is rare in myopia. Recessive and sex-linked is also a peculiar form of myopia which is associated with night-blindness, and will be described later in connexion with that anomaly. From Vogt's clinic we get, moreover, a genealogical tree showing the recessive hereditary transmission of high-grade myopia in association with high-grade nyctalopia (night-blindness). See below, p. 255.

Reference must also be made here to *detachment of the retina*, which is unfortunately an occasional sequel of high-grade myopia. Whereas in most cases the elongation of the globe which gives rise to short-sight ceases when the general arrest of growth takes place with the attainment of maturity, in some instances this elongation continues with disastrous results, the retina undergoing partial or complete detachment, with consequent partial or complete blindness. In a family reported on by Bogatch, among 11 members affected by extreme myopia, there were 7 in whom detachment of the retina in one or both eyes supervened. In such persons the short-sight may be presumed to be genetically distinct from the ordinary kind.

Like short-sight, *long-sight*, technically known as *hypermetropia* or *hyperopia*, has been shown to be hereditary. In this condition the axis of the globe is too short, so that when the eye is at rest (i.e. unaccommodated) the parallel rays from distant objects are focussed behind the retina instead of on it, with resulting indistinctness of vision. To secure clear vision even of distant objects the hypermetrope has therefore to make the effort of accommodation which in persons having normal refraction need be made only for near vision, and in high degrees of the defect the continuous eye-strain may produce grave consequences. Speaking generally, hyperopia appears to be dominant as compared with emmetropia (the condition in which refraction is normal). This coincides with the results of enquiries made by Jablonski, who found that a shorter axis of the globe is wholly or partly dominant as against a longer axis. A family tree illustrating the incidence of hyperopia is given in Fig. 39.

Likewise hereditary in the main is the error of refraction known as *astigmatism.* In this condition the anterior surface of the cornea is more strongly curved in one axis than in another, instead of being, as it should be, perfectly spherical. The result is that when the rays refracted through one axis are being focussed on the retina, the rays refracted through another axis are being focussed in front of or behind it. Thus the definition of all objects is impaired, no matter whether the eye is accommodated for near vision or not. Steiger's exhaustive researches enabled him to prove that astigmatism is hereditary. In one family Spengler was able to trace the occurrence of astigmatism for five successive generations, and to show that the axis and the degree of the astigmatism were the same throughout. As a rule, astigmatism seems to be a dominant character, but we may suspect that the heredity factors which induce this condition are not of one kind alone.

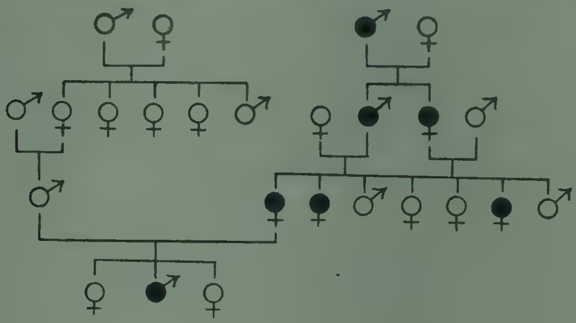

FIG. 39.
Hyperopia, after Jablonski.

It would, of course, be an error to suppose that only the morbid deviations from the normal condition of refraction are hereditary. Normal refraction, emmetropia, is likewise hereditarily determined, a great number of genes being jointly at work in its production. When one of these genes undergoes a change (mutation), a hereditary anomaly of refraction originates, and we should obviously expect that there would be many different kinds of such anomalies.

We may compare the heredity of the ratio between the length and breadth of the globe of the eye with that of the similar ratio between the length and breadth of the skull—and, indeed, of the body in general. In these latter cases also there are hereditary differences of shape which cannot be described as simply dominant or simply recessive. Nor, indeed, could we expect anything of the kind in the domain of non-morbid differences (see above, pp. 220–1). Still, there can be morbid hereditary trends endowed with a simple heredity, and manifesting themselves strongly, in the shape of the head and in that of the body. For instance, there is a recessive hereditary morbid dwarfing, which may be compared with recessive hereditary high-grade myopia.

We find geographical differences in the shape of the globe, just as in the shape of the head and of the body. In Anglo-Saxon countries short-sight is much less common than in continental Europe. According to Czellitzer, in Schleswig-Holstein before the war among the one-year-volunteer recruits there were only 24–5 % of myopes, whereas in southern Bavaria there were 41–8 %. That may depend upon the circumstance that in north-western Europe the population has been mainly formed by a mingling of various dolicocephalic races, whereas in central Europe dolicocephals and brachycephals have been crossed. This implies the theory that anomalies of refraction may sometimes be the outcome of racial mixings. A particular curvature of the cornea which may produce normal vision when conjoined with a particular length of the globe, may produce a moderate degree of short-sight when conjoined with a longer globe; and, conversely, the length of axis which in conjunction with this particular curvature of the cornea produces short-sight, may in conjunction with a different curvature of the cornea produce emmetropia. Slight differences in the shape of the body have in most cases no pathological significance; but in the eye, whose supreme function it is to produce clearly defined images on the retina, even a trifling elongation of axis will cause short-sight. On the other hand, a very slight shortening in the axis of the globe is devoid of pathological importance. It would seem, even, that in most people when the eye is at rest the images of distant objects are focussed in a plane that lies just a little behind the retina. Since a very slight increase in the curvature of the lens (accommodation) then suffices to secure accurate focussing upon the retina, a slight degree of long-sight is not, as is a slight degree of short-sight, necessarily morbid.

Squint, technically known as *strabismus*, is often associated with errors of refraction, and especially with hyperopia. Accommodation for near vision is normally associated with convergence of the axes of the eyes, which are both turned slightly inwards to be directed towards the near object. The association of accommodation with convergence is thus prearranged in the nervous mechanism. Since, in hyperopes, there is a persistent effort of accommodation (even when they are looking at distant objects) it is natural that in them there should be a marked tendency to the development of a persistent and exaggerated convergence of the axes, and therefore to the onset of an internal squint. This is the commonest form of squint, the "strabismus convergens concomitans" associated with hyperopia. It must not, however, be supposed that squint is an invariable accompaniment of "long-sight". Contributory causes of strabismus are a weakness in the capacity for fusing the images formed by the two eyes and a weakness in the ocular muscles or in their innervation. As Clausen especially has insisted, we must not expect to find a monomeric heredity in squint. The heredity factors which induce squint would seem to be mainly recessive. Clausen[1] and Czellitzer,[2] who have collected a number of genealogical trees showing the incidence of the affection, are agreed upon this matter.

Czellitzer, whose material extends to 306 families, found that the squinting children of healthy parents had 14·0 ± 1·2 % of squinters among their brothers and sisters; but that when one of the parents also squinted, the proportion of squinters among the brothers and sisters was 29·5 ± 4·9 %. Classing internal squint and external squint separately, we find that in the former case the respective proportions (according as neither parent squinted or one parent squinted) were 15·1 ± 1·7 % and 40·0 ± 6·7 %, and in the latter case 7·2 ± 2·0 % and 11·0 ± 7·0 %. The parents of squinting children were closely related by blood in the proportion of 6·0 ± 1·5 % as contrasted with a proportion of 0·62 % of consanguineous marriages in the general population of Berlin (whence most of Czellitzer's material was derived). Unfortunately, Czellitzer has not published his genealogical trees showing the family incidence of squint.

Fig. 40 shows a genealogical tree[3] which may be accounted for as the expression either of recessive or of dominant heredity. It is obvious that the hereditary determination of squint is not of only one kind. Fig. 41 shows a genealogical tree which speaks strongly in favour of a dominant heredity. Inasmuch as hyperopia is usually a dominant character, it would seem probable that dominant heredity factors are likewise concerned in the production of the accompanying squint—even though hyperopia is far from being always attended by strabismus.

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[1] W. Clausen and J. Bauer, Beiträge und Gedanken zur Lehre von der Vererbung des Strabismus concomitans, "Zeitschr. für Augenheilkunde", vol. L, nos. 5 and 6, 1923.

[2] A. Czellitzer, Wie vererbt sich Schielen! "Archiv für Rassen- und Gesellschaftsbiologie", vol. XIV, no. 4, 1923.

[3] A. Vogt, Ueber Vererbung von Augenleiden, "Schweizerische Mediz. Wochenschr.", nos. 7 and 8, 1923.

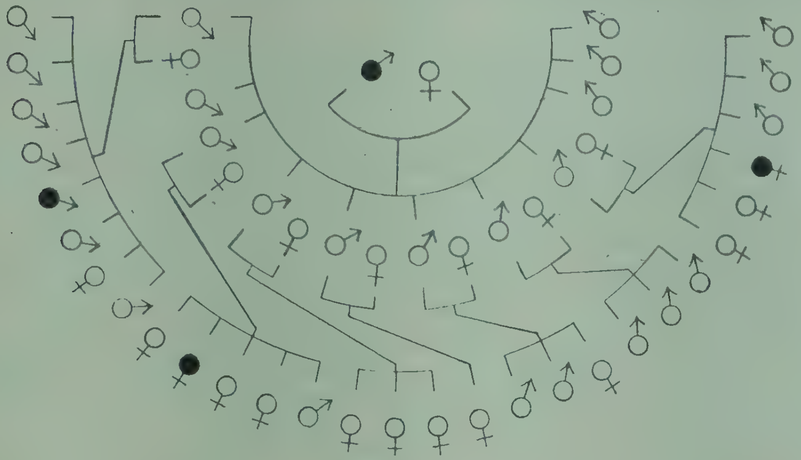

FIG. 40.
Internal squint associated with hyperopia, after Vogt.

Squint may also be an outcome of a unilateral weakness, the asthenic eye tending to diverge more often from the axis of vision.

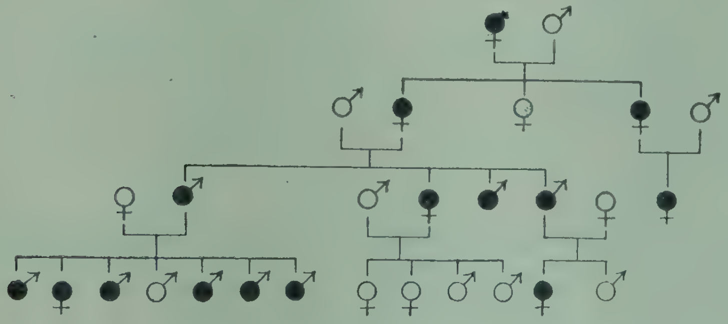

FIG. 41.
Internal squint associated with hyperopia, after Clausen and Bauer.

Peters has written about the inheritance of this form of strabismus.[1] Presumably many of the defects of the eye muscles and nerves which eventuate in squinting are hereditary.

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[1] Ueber die Bedeutung der Erblichkeit des Schielens, "Klinische Monatsblätter für Augenheilkunde", vol. LXVIII, 1922.

We owe to Sicherer [1] a report of considerable theoretical interest, relating to a family in which for four generations all the boys had hyperopia restricted to the left eye and all squinted. This family history deserves the most careful study, for it might help to throw light on the problem of the inheritance of characters which appear on only one side of the body.

Nothing is known as to environmental causes of squint, and it is probable that heredity is the exclusive explanation of the anomaly. But in a good many squinting children a spontaneous cure takes place as they grow up and this might be expected seeing that hyperopia is at its worst in early childhood and tends to diminish with advancing years. In other cases squint may be cured by operation. More than 2 % of school children squint, but only about 1 % of adults. The liability to squint is approximately the same in the two sexes.

Hereditary tremor of the eyes (*nystagmus*) occurs, not only as an accompaniment of albinism, but also as an independent anomaly. Usually there is associated amblyopia. In certain families the heredity of nystagmus is unmistakably dominant, and in the members of such families the tremor of the eyes is apt to be accompanied by kindred twitching movements of the head. W. Nicoll [2] has described a family in which 21 members, belonging to 6 generations, suffered from such nystagmus.

In other families, however, nystagmus shows a recessive sex-linked heredity, and this form is not usually associated with spasmodic twitching of the head. Engelhard has described a family in which 19 of the male members and one of the females suffered from nystagmus.[3] We owe to G. D. Hemmes an exhaustive study of hereditary nystagmus.[4]

Another hereditary anomaly of the movements of the eye and its accessory parts is inability to raise the upper lid, this being technically known as *ptosis*. Only by wrinkling the forehead is the sufferer from ptosis able to a small extent to lift the upper lid of the affected eye, the muscle which has the special function of raising the lid being paralysed. The inheritance of this trouble can often be traced for a number of generations, and it is manifestly dominant. In a family described by H. Briggs,[1] 64 of the members suffered from ptosis. Among the children of the patients, 64 were affected and 60 were free from the trouble, which closely corresponds to the theoretical ratio of 1 : 1.

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[1] Vererbung des Schielens, "Münchener Medizinische Wochenschrift", 1907, p. 1231.

[2] A Pedigree of Hereditary Nystagmus, "The Ophthalmoscope", 1915.

[3] Eine Familie mit hereditärem Nystagmus, "Zeitschr. für die gesamte Neurologie und Psychiatrie", vol. XXVIII, nos. 4 and 5, 1915.

[4] Over hereditairen Nystagmus (Dissertation), Wageningen, 1924.

In most cases of hereditary ptosis, the palpebral fissure is abnormally narrow, and in many cases there is an *epicanthus*—a fold of skin connecting the upper lid and the lower across the inner angle of the eye. In some families the two anomalies are always associated, as we see in a family history given by Steinheim[2] (14 persons in 5 generations), and learn from another instance described by C. H. Usher[3] (18 persons in 4 generations). Here the two anomalies must be caused by the same morbid taint. But in other families we find ptosis without epicanthus, and in yet others epicanthus without ptosis. Epicanthus existing by itself can hardly be regarded as having a pathological significance, and cannot always be distinguished from the racial character known as the Mongolian fold (see above, p. 131). A remarkable genealogical tree published by E. Meirowsky[4] shows that this anomaly taken by itself can manifest a dominant heredity, but such dominance is not invariable. In 150 pairs of mon-oval twins, Meirowsky found epicanthus in 11 instances in both members of a pair; in 150 pairs of di-oval twins, epicanthus was in 4 instances present in both members of a pair, and in 3 instances in only one member of a pair.

There is sometimes a hereditary paralysis of all the muscles of the eye, so that the organ cannot be moved. Such a paralysis is known as *ophthalmoplegia*. Congenital ophthalmoplegia has in some families occurred generation after generation only in males, but in others in females as well. We also have records of families in which several members, though born with eyes capable of being moved in normal fashion, became during youth gradually affected with ophthalmoplegia. This trouble seems to be analogous to the hereditary atrophy (presently to be described) which sometimes affects the larger muscles.

Commoner are hereditary anomalies of the *lachrymal sac*. Abnormal narrowness or even complete imperviousness of the lachrymal canal is sometimes met with in several members of a family. Chronic suppurative inflammation of the lachrymal sac is also in some instances a family ailment, being then presumably dependent upon hereditary impairment of the power of resisting the exciting causes of this particular local inflammation.

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[1] Hereditary congenital Ptosis, "AmericanJournal of Ophthalmology", vol. II, 1919.

[2] Epicanthus mit Ptosis, "Zentralblatt für prakt. Augenheilkunde", vol. XXII, 1898.

[3] A Pedigree of Epicanthus and Ptosis, "Annals of Eugenics", vol. I, nos. 1 and 2, 1925.

[4] Kleinere Mitteilungen zur Erblichkeitslehre, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XVII, no. 4, 1926.

The same remark applies to chronic inflammation of the margins of the eyelids (*blepharitis ciliaris*), which is likewise a family complaint. *Spring catarrh* (vernal conjunctivitis), an inflammation of the conjunctiva which tends to recur every spring and is due to a morbid sensibility of the membrane to sunlight, appears to be the outcome of a hereditary predisposition running in particular families.

A rare anomaly termed *distichiasis*, in which, besides the normal eyelashes, a second row appears on the margin of the lid in the region of the Meibomian glands, exhibits according to Scily a recessive heredity.[1]

An abnormally large cornea (*megalocornea*), owing to the presence of which the eye appears to be of an exaggerated size, has been observed in a number of families as a recessive sex-linked hereditary character. This anomaly can, however, hardly be said to have a pathological significance.

Hereditary corneal opacity, appearing as a family trouble, was first observed by B. Fleischer,[2] and subsequently by other ophthalmologists. At birth, in such persons the cornea is transparent, but during the years of growth cloudy areas make their appearance, and in course of time the opacities become so dense that the power of vision is almost entirely lost. The form of the opacities differs in different families; sometimes they are punctiform, sometimes they appear in lines, and sometimes they are patchy; but in members of the same family they are always alike. There would seem, then, to be several different dominant anomalies. In the case of four families affected with this trouble Fleischer was able to show that they had had a common ancestor seven generations back.

Abnormal smallness of the eye (*microphthalmia*) has not infrequently been observed in several brothers and sisters; the parents have usually had normal eyes, but in some of the instances they were consanguine. Complete absence of the eyes (*anophthalmia*) has also been observed in several brothers and sisters. Microphthalmia and anophthalmia may likewise occur simultaneously in various members of the same generation in one family. We may presume that these anomalies are the outcome of various recessive heredity factors which must cooperate to produce them, the gravity of the malformation depending on the extent of the cooperation.

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[1] Ueber Haarbildung in den Meibomschen Drüsen, etc., "Klinische Monatsblätter für Augenheilkunde", vol. LXX, 1923.

[2] Ueber familiäre Hornhautentartung, "Zentralblatt für Augenheilkunde", vol. LIII, 1905.

In a family reported on by W. H. Ash,[1] microphthalmia recurred during three generations as a recessive sex-linked character. O. Purtscher[2] traced the association of microphthalmia with myopia in four generations of a family; here there seems to have been a peculiar dominant (or intermediate) anomaly.

Lack of the iris (*aniridia* or *irideremia*) occurs as a dominant hereditary malformation. W. Clausen has published a genealogical tree, reproduced as Fig. 42, showing the family incidence of this malformation.[3] In many such families there are also persons who exhibit no more serious a malformation than a cleft iris (*coloboma iridis*). It would seem, then, that the same heredity factor may produce effects of varying gravity according to the way in which it is linked with other factors, causing now a mere coloboma and now again complete aniridia.

Coloboma iridis has in a number of families been traced for many generations, so that we may suppose its heredity to be dominant or intermediate. In homozygotics there can be little doubt that the same heredity factor would cause a graver defect of development, presumably an entire absence of the iris (aniridia). But since there are families in which some members have aniridia and others coloboma, whereas there are other families in which cases of coloboma occur in the absence of cases of aniridia, there must be distinct heredity factors at work in the respective families, factors whose effects are quantitatively different. In two families, coloboma occurred for five generations in each, but there were no instances of complete aniridia.

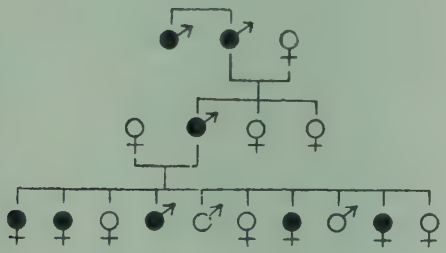

FIG. 42.
Aniridia.

Congenital displacement of the lens (*ectopia lentis*) has several times been traced through four generations, in two families through five generations, and in one family actually through six generations. Here dominance is unmistakable.

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[1] Hereditary Microphthalmia, "British Medical Journal", 1922.

[2] Angeborene Missbildung bei einem Brüderpaar, "Archiv für Ophthalmologie", vol. CV.

[3] Aniridia congenita und Heredität, "Klinische Monatsblätter für Augenheilkunde", vol. LXVII, 1921.

There is also a form of displacement of the lens associated with displacement of the pupil, and H. W. Siemens regards this as recessive.[1]

It is remarkable to find ectopia of the lens and ectopia of the pupil associated with valvular disease of the heart. This occurred in the members of a family described by J. Strebel.[2] (See below, p. 373.)

A. Vogt has given an account of a family in 18 members of which a dislocation of the lens occurred after they had grown up. A dominant heredity factor must have been at work. The anatomical cause of the trouble was the atrophy of the suspensory ligament of the lens (the zonule of Zinn).[3]

Hereditary opacity of the lens (*cataract*) is extremely important, both on account of the gravity of the resulting disturbance of sight and on account of its frequency. What is ordinarily spoken of as "senile cataract"—the cataract which only makes its appearance in persons well advanced in years—was apt until recently to be looked upon as exclusively due to old age. Nevertheless there are plenty of old people of eighty years and more who show not a sign of cataract, whereas in others the trouble begins in middle age. External causes, such as radiant heat, among furnace workers for instance, may favour the onset of cataract; but such noxious influences act far more quickly and injuriously upon some persons than upon others.

Heredity is, of course, most conspicuous in cases of congenital cataract. In this form of the disorder we usually find that the lens is not wholly obscured, the cataract being sometimes nuclear, sometimes cortical, and sometimes lamellar. The heredity of congenital cataract is usually dominant.

In any particular family the age at which cataract begins is fairly constant. We find some families in whose members cataract appears in early childhood; others in whose members it begins at puberty; and yet others in whose members the trouble dates from middle life. The heredity seems in all cases to be dominant. Although in families affected with this taint we sometimes find that, notwithstanding the general dominance of the morbid heredity, certain members appear to have remained immune, the immunity has presumably been

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[1] Ueber die Aetiologie der Ectopia Lentis et Pupillae, etc., "Archiv für Ophthalmologie", vol. CIII, 1920.

[2] Korrelation der Vererbung von Augenleiden, etc., "Archiv für Rassen- und Gesellschaftsbiologie", vol. X, no. 4, 1913.

[3] Dislocatis lentis spontanea als erbliche Krankheit, "Zeitschr. für Augenheilkunde", vol. XIV, 1905.

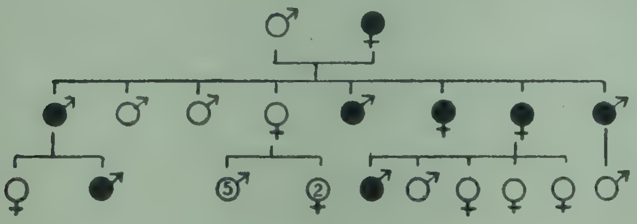

FIG. 43.
Congenital nuclear cataract, after Nettleship (part only of the family tree is given).

apparent only. The probable explanation is that those who escaped did not live long enough for their tendency to cataract to become active (cf. Fig. 44).

Clausen writes as follows regarding the hereditary transmission of cataract: "Cataract being hereditary in certain families, we generally find the same form of cataract in the various members of

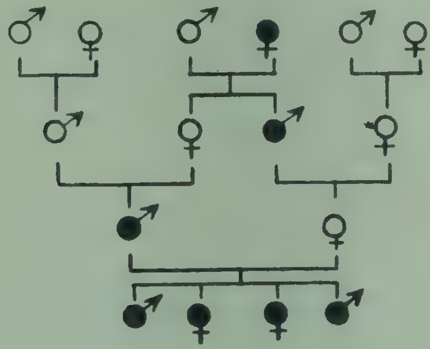

FIG. 44.
Cataract, after Grote.

a particular family. Sometimes we have to do with a congenital cataract in which the lens is totally obscured; sometimes with a cataracta zonularis, coralliformis, centralis, stellata, fusiformis, coronaria, etc., as the case may be. There is also a hereditary form of cataract which only begins to appear when those who have the taint are well on in years (being thus analogous to progressive senile cataract), but in which not infrequently we observe the remarkable phenomenon of *anticipation*. This means that as we pass down the generations the cataract tends to appear at an ever earlier age. Such an anticipation was especially marked in a cataract family studied by G. Norrie.[1] In the first generation the cataract began in old age; in the second generation at 40; in the third generation at 30; in the fourth generation before the age of 7; and in the fifth generation, very soon after birth.

I should like to express my doubts concerning this doctrine of "anticipation". In view of our general knowledge of biological processes, it seems to me most unlikely. In part, at any rate, a statistical illusion will account for apparent "anticipation".[2] Since blind persons or those whose sight is gravely impaired seldom marry, persons with a hereditary tendency to cataract will not be likely to have children unless they are among the patients in whom for one reason or another the cataract does not appear until they are well on in years. The parental cataractous patients in our genealogical trees were therefore singled out as persons in whom cataract does not appear before old age; but in the living generation a reverse kind of "selection" is at work, for only such persons are found to be affected as have developed cataract comparatively early, whereas those of their brothers and sisters in whom cataract will perhaps appear subsequently are for the nonce described as healthy. Thus in many families we shall get ostensible "anticipation" although there is no valid biological reason for supposing that anything of the kind is taking place.

Of course an anticipation as extensive as that described by Norrie could not be accounted for in any such way. His data are probably now out of reach of the possibility of a critical re-examination, and we must await the careful scrutiny of any future instances of an apparently similar anticipation. It is certainly remarkable that no such instances have been recorded, no cases of extreme anticipation, since the days when the achievements of contemporary genetics became generally known. If such a sequence as that described by Norrie were to be frequently observed, we might explain it on the supposition that a taint which, acting heterozygotically, had induced senile cataract, was subsequently able, acting homozygotically, to induce cataract in youth. In the case of recessive troubles (such as hereditary ataxia) it is known that a hereditary taint may be transmitted heterozygotically for several generations, and may at length manifest itself homozygotically in several members of a family. Something of the sort might, of course, likewise occur in the case of dominant troubles, and this would result in the phenomenon of marked anticipation.

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[1] Arvelighed of grau Star, "Ugeskrift for laeger", Copenhagen, 1896.

[2] W. Weinberg seems to have been the first to notice this. See his paper, Auslesewirkungen bei biologisch-statistischen Problemen, "Archiv für Rassenbiologie", vol. X, nos. 4 and 5, 1914.

Congenital cataract is one of the most important causes of *congenital blindness*. Among 1300 blind persons, Hirsch found that in 111 (nearly 10 %) congenital cataract was the cause of the blindness.[1] Anophthalmia and microphthalmia, taken together, come next as cause of congenital blindness. Of young blind persons, about one fourth will be found to have been born blind, and congenital blindness is almost always hereditarily determined. Acquired blindness, on the other hand, is predominantly due to other causes than heredity—such causes as gonorrhoeal infection of the conjunctiva during birth, accident, etc. But among the 30,000 blind persons in Germany several thousand certainly owe their affliction to morbid inheritance.

A fairly frequent cause of the onset of blindness in middle life is the disease known as *glaucoma*, in which a rise of intra-ocular tension leads to atrophy of the retina. We distinguish between acute or inflammatory glaucoma, which is attended by severe pain and may cause loss of sight within a few days, and chronic or simple glaucoma, in which there is comparatively little pain occurring at odd times, and the onset of blindness is gradual. In members of the same family, glaucoma is usually of the same type.

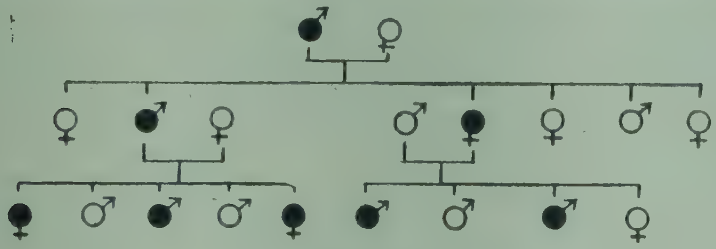

FIG. 45.
Inflammatory glaucoma, after Howe.

The genealogical trees hitherto published illustrating the family incidence of glaucoma suggest that the heredity is mainly dominant. The hereditary course is, indeed, occasionally interrupted; but we may suppose that this is because casual exciting causes are requisite to make the hereditary predisposition to glaucoma active (closure of the canal of Schlemm, through which there normally occurs an outflow of fluid from the interior of the eye).

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[1] A. Czellitzer, Augenfehler, in Grotjahn and Kaup's Handwörterbuch der socialen Hygiene, Vogel, Leipzig, 1912.

In certain families, glaucoma may develop quite early, at an age ranging from 15 to 20. In one family the incidence of this "juvenile glaucoma" could be traced through five generations. "Anticipation" has been recorded in the case of this malady likewise. A. von Graefe[1] tells us that in certain families the parents or grandparents had become affected during the sixth decade of life, whereas in the younger generation the disease appeared during the fourth decade. This amount of "anticipation" can certainly be explained by an unintentional statistical selection of the kind described above (p. 244).

Since glaucoma has sometimes been observed in brothers and sisters when no earlier family history of the complaint was obtainable, we must infer that the heredity may occasionally be recessive. We still lack adequate information concerning the frequency of consanguineous marriages among the parents of glaucoma patients. One indication in favour of a recessive heredity is furnished by the circumstance that glaucoma is markedly less common among Gentiles than among Jews, in whom consanguineous marriages are comparatively frequent.

In the year 1905 among the Jews of Prussia the proportion of blind persons was 71 per 10,000, whereas the proportion in the population at large was only 56. This excess of blindness among Jews must be wholly the outcome of morbid heredity, especially in view of the fact that Jews are particularly careful of their health and follow few of the occupations in which injuries to the eye are especially likely to occur. But we are not yet in a position to say whether the greater frequency of hereditary blindness among Jews depends upon the greater frequency of consanguineous marriages, or whether certain heredity factors of blindness are more prevalent in the Jewish stock.

Glaucoma, especially of the inflammatory type, is comparatively frequent among psychopaths. There are certain families in whose members the onset of glaucoma is usually associated with symptoms of mental depression and with precordial anxiety—this showing how various bodily and mental disturbances may be induced by one and the same gene.

Somewhat akin to glaucoma is the condition known as *hydrophthalmus*, which is likewise hereditary. This distension of the eyeball with watery effusion is due to the absence of the before-mentioned canal of Schlemm, and it causes loss of sight in early childhood. A number of family trees collected by Paula Werth indicate that in most cases the heredity is recessive. Where the incidence in particular families was marked, nearly 50 % of the patients were the offspring of consanguineous marriages.

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[1] Beiträge zur Pathologie und Therapie des Glaukoms, "Archiv für Ophthalmologie", vol. XV, 1869.

Optic neuritis and *optic-nerve atrophy* are a frequent cause of blindness, and the hereditary course of this trouble is well known. It usually comes on during the second, third, or fourth decade of life. To the accompaniment of inflammatory phenomena in the optic nerve, in the course of a few days the power of vision may be completely lost in the centre of the visual field, the part upon which clear vision depends. Often, however, the course of the malady is slower. Some power of vision is usually left in the periphery of the visual field. In any particular family, the course of optic-nerve atrophy is usually much the same. As a rule in such families the men only are affected. The heredity is in most instances recessive and sex-linked.

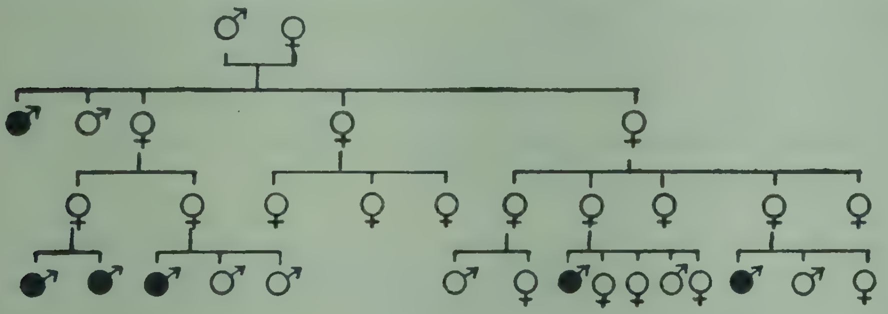

FIG. 46.
Optic-nerve atrophy, after Hensen.[1]

We have records of a great number of families liable to optic-nerve atrophy. In several instances, the malady has been traced through four generations; twice, through five generations; and once, through six generations. In a family described by B. Fleischer and W. Josenhans,[2] seventeen of the males were affected in four generations, but in this family, as usual in such families, the females remained immune. Still, we have a few family trees showing the incidence of the malady in the female members as well; and, moreover, these are instances in which the father was healthy, so that we have not to do here with homozygotically affected women, such as there may be in the case of sex-linked maladies which are wholly recessive. A peculiarly instructive genealogical tree showing sixteen males and two females in one family affected with this state, has been published by P. I. Waardenburg.[1] Since these women both had healthy fathers, and since some of their sons were likewise healthy though others were affected, they cannot be regarded as homozygotically diseased. This signifies, however, that the sex-linked tendency to optic-nerve atrophy cannot be wholly recessive (at least in certain families), but that in exceptional circumstances it can manifest itself heterozygotically in females as well as in males. The taint thus shows itself to be incompletely or irregularly recessive and sex-linked. This type of heredity is transitional towards dominant sex-linked heredity, but stands much nearer to recessive sex-linked heredity.

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[1] Hensen, Ueber Neuritis optica hereditaria, "Klinische Monatsblätter für Augenheilkunde", vol. LIX, no. 3, 1917.

[2] Ein Betrag zur Frage der Vererbung der familiären Sehnervenatrophie, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XIII, nos. 2–4, 1920.

Another kind of optic-nerve atrophy has been observed in families as an accompaniment of hereditary ataxia[2] (see p. 413 et seq.). The heredity was apparently dominant. Optic-nerve atrophy is also fairly common in cases of the non-hereditary ataxia (tabes dorsalis) which occurs as a sequel of syphilis. Behr[3] has described a peculiar form of hereditary optic-nerve atrophy which manifests itself already during childhood. Congenital optic-nerve atrophy or aplasia (defective formation) has in sixteen instances been observed by Clausen in children of the same parents, once affecting five out of eight such children. The malady is presumably recessive. This form of hereditary blindness is on the same footing as hereditary deaf-mutism.

Nearly 4 % of all cases of blindness are stated by Czellitzer to be the outcome of hereditarily determined atrophy of the retina (*retinitis pigmentosa*). The trouble begins in early youth with a restriction of the visual field, which is slowly progressive, until at length, after some decades, central vision, too, is finally lost. Retinal atrophy, or retinitis pigmentosa, is a clinical name for a malady of which there exist several kinds whose symptoms and course are similar though they are biologically different, as is shown by their varying heredity. The commonest of them is a recessive form.

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[1] Beitrag zür Vererbung der familiären Sehnervenatrophie, "Klinische Monatsblätter für Augenheilkunde", vol. LXXIII, 1924, p. 619.

[2] Wilbrand and Saenger, Handbuch der Neurologie des Auges, vol. V, Erkrankungen des Optikusstammes, Bergmann, Wiesbaden, 1913.

[3] Die komplizierte hereditär-familiäre Optikusatrophie des Kindesalters, "Klinische Monatsblätter für Augenheilkunde", vol. XLVII, 1909, p. 138.

Associated with the recessive character of the heredity is the fact that persons suffering from retinal atrophy are with remarkable frequency the offspring of consanguineous marriages—to the extent, indeed, of nearly one third. In a genealogical tree recorded by F. M. Boehm[1] and reproduced in Fig. 47, we see that, two brothers having married two sisters, among the offspring of these unions there was in each case one person affected with retinal atrophy. In this instance, the disease of the retina was associated with atrophy of the choroid (the vascular coat of the eye). The same association has been noted in certain other families. There are yet other families in which, in certain members, retinal atrophy is regularly associated with deaf-mutism. Manifestly, therefore, there are several idiotypically different kinds of recessive retinal atrophy.

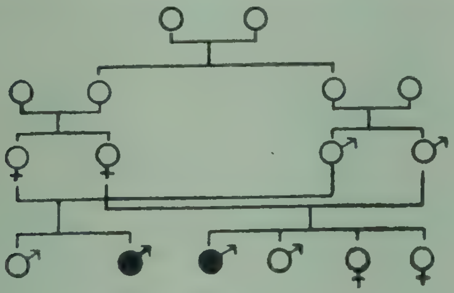

FIG. 47.
Recessive retinal atrophy, after Boehm.

Certain families have, moreover, been observed in which there is a dominant tendency to retinal atrophy. See, for instance, Fig. 48.

In one family described by Nettleship, the retinal atrophy could be traced through six generations, affecting thirty-eight persons in all. By other observers, this malady has in several instances been kept under observation for three generations, in one instance for four generations, and in one instance for five. But dominance is the exception. In the great majority of cases, retinal atrophy is determined by recessive heredity factors.

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[1] Ueber einen eigentümlichen Fall von Retinitis pigmentosa mit Atrophie der Aderhaut, "Klinische Monatsblätter für Augenheilkunde", vol. LXIII, no. 2, 1919.

Finally I should mention that there is also a recessive sex-linked kind of retinal atrophy, as is shown in Fig. 49.

In the *Treasury of Human Inheritance* mentioned on p. 223 there are reproduced no less than 366 genealogical trees showing the family incidence of retinal atrophy and kindred disorders, and in five instances the heredity was apparently dominant. In 27 % of the cases, the parents had been of near kin. Eliminating the instances of dominant heredity, we find that in the simple recessive heredity consanguineous marriage has played a part in at least 30 %. In the Netherlands, according to P. A. de Wilde,[1] the total number of extant cases of retinal atrophy is about 200, this representing a frequency of 1 : 20,000. The proportion of males to females among the patients is about 3 : 2. This excess of males must be an expression of the prevalence of the recessive sex-linked form.

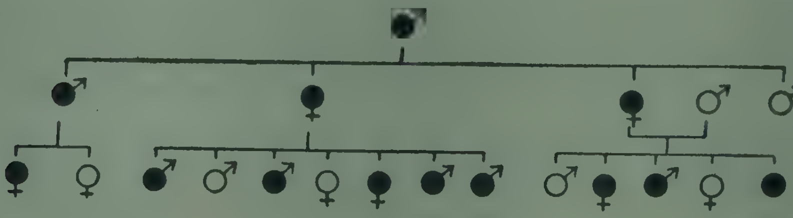

Fig. 48.

Dominant retinal atrophy, after Nettleship (part only of the family tree is given).

Whereas in ordinary retinal atrophy it is the area of central distinct vision which is longest preserved, we meet with hereditary affections of the eye in which this very region, that of the macula lutea or yellow spot, is the first to suffer. In different families, different forms of this *macular atrophy* have come under notice, distinguished from one another by the age at which the malady appears—the age-incidence recurring with remarkable accuracy in

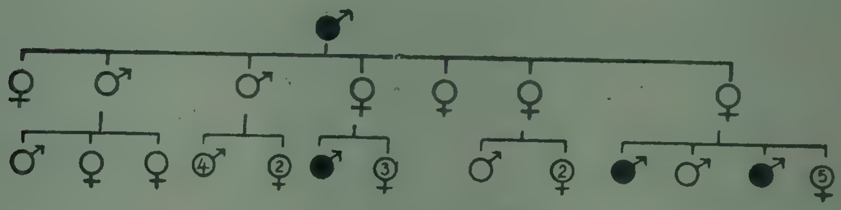

Fig. 49.

Recessive sex-linked retinal atrophy, after Nettleship (part only of the tree is reproduced).

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[1] Verwandtschaft und Erblichkeit bei Taubstummheit und Retinitis pigmentosa (Dissertation), Amsterdam, 1919.

the respective members of any one family.[1] In one family, the loss of central vision will occur in early childhood; in another family, at the age of puberty; in another, during the third decade; and in yet another, not until the sixth decade. Here, it would seem, we have to do with various troubles having a recessive heredity which is biologically different in each case. In one of the genealogical trees published by Behr, in which macular atrophy could be traced in seventeen persons during four generations, the heredity was certainly dominant, although the taint was twice transmitted by healthy males.

To the group of macular atrophies we can also assign the cases in which blindness is associated with idiocy (amaurotic idiocy). This will be described in the subsection on mental disorders.

Vogt has observed cases of a congenital lack of the macula lutea in which the heredity appeared to be recessive sex-linked.

Night-blindness is an anomaly in which the existence of an abnormal structure of the retina can only be inferred from the failure of the capacity to see as well as normal people do in a faint light. Though comparatively blind in the deep twilight conventionally called "darkness", such persons can see as well as any one else in the broad light of day. The British ophthalmologist E. Nettleship[2] has published the genealogical tree of a night-blind family in which the cases of night-blindness were noted for nine generations, the malady affecting 135 persons out of a total number of 2116. This is the most extensive family tree hitherto constructed to illustrate morbid heredity. In the case of every one of these 135 night-blind persons, the morbid taint can be traced back in unbroken series to a night-blind butcher named Nougaret who was born in the year 1637 at Vendemian near Montpellier. Every night-blind member of this kinship had at least one night-blind parent; in one instance, both the parents were night-blind, and they had two night-blind daughters. If both parents were free from night-blindness, none of their children suffered from the malady. The taint is therefore dominant. I will illustrate the foregoing with two extracts from the Nougaret tree.

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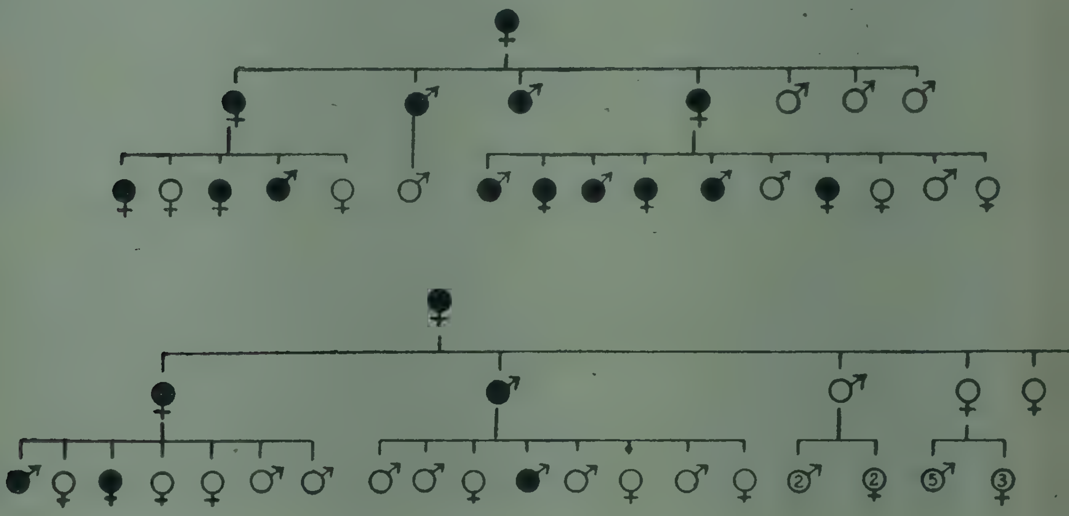
[1] C. Behr, Die Heredodegeneration der Makula, "Klinische Monatsblätter für Augenheilkunde", vol. LXV, 1920.

[2] A History of Congenital Stationary Night-Blindness in Nine Consecutive Generations, "Ophthalmological Review", 1907.

In the upper of the two following extracts from the tree, we see the majority of individuals affected, whereas in the lower extract only a minority suffer. But this difference is not the expression of any biological factor at work. In accordance with the mendelian law, we must expect that, on the average, half the children would be night-blind and half of them normal. Any one child, therefore, has an equal chance of being night-blind, and each of his brothers and sisters has the same chance. Consequently, in accordance with the laws of probability, in so extensive a kinship we should expect to find branches in which many individuals would suffer and other branches in which few would suffer. The two extracts from the Nougaret tree given in Figs. 50 and 51 were specially chosen to illustrate that the offspring of any particular pair could not be expected to show mendelian ratios in the morbid inheritance, although the general distribution of the taint conforms satisfactorily to the mendelian law.



FIGS. 50 and 51.

Dominant night-blindness, extracts from the Nougaret genealogical tree, after Nettleship.

In Fig. 52, I give another extract from the Nougaret tree which shows the uninterrupted inheritance of night-blindness for nine generations.

Very striking in the Nougaret family tree is the fact that among the children of night-blind individuals, 134 were night-blind and 246 were healthy. Siemens suggests that some of the children may have erroneously been supposed to be healthy, especially when they died young; but he considers that the whole difference between the actual figures and the theoretically correct ratio of 190 : 190 cannot be accounted for in this way. I would myself suggest as a possible explanation that the spermatozoids conveying the tendency to night-blindness may have had less capacity for effecting fertilisation of the ovum than those not conveying this taint. Certainly we find that the excess of healthy children over night-blind children is greater among the offspring of night-blind fathers, than among the offspring of night-blind mothers. Still, Siemens' suggested explanation may in part account for the fact, especially since the genealogical tree was partly compiled from data furnished by non-medical persons.

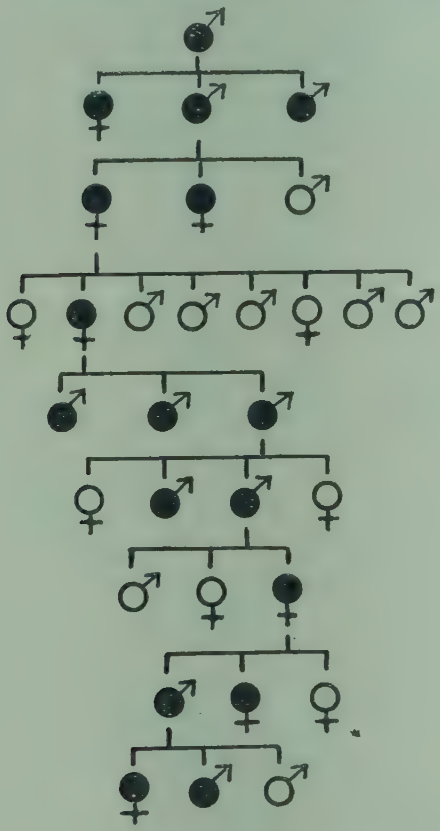


FIG. 52.

Dominant night-blindness, extract from the Nougaret genealogical tree, after Nettleship.

In addition to this dominant night-blindness, there is another kind which, in contradistinction to that previously described, is regularly associated with short-sight. We have a number of genealogical trees showing the incidence of this latter form, and it appears from these that it is recessive sex-linked. Very carefully compiled trees illustrating the heredity of this malady have been published by W. Kleiner[1] and H. Varelmann.[2]

This genealogical tree can also be used to illustrate the fact that short-sight may be the expression of a recessive sex-linked taint.

A third kind of night-blindness which is associated with high-grade myopia and shows a simple recessive heredity, has been observed in Switzerland by Vogt. In Fig. 54 I reproduce a family tree published by V. J. Gassler,[3] a pupil of Vogt. It is instructive as regards the heredity of recessive taints in general.

In Japan, simple recessive night-blindness seems to be commoner than among ourselves, being there known as Oguchi's disease.[4] We do not know as yet whether this malady is identical with the recessive night-blindness discovered in Switzerland by Vogt.

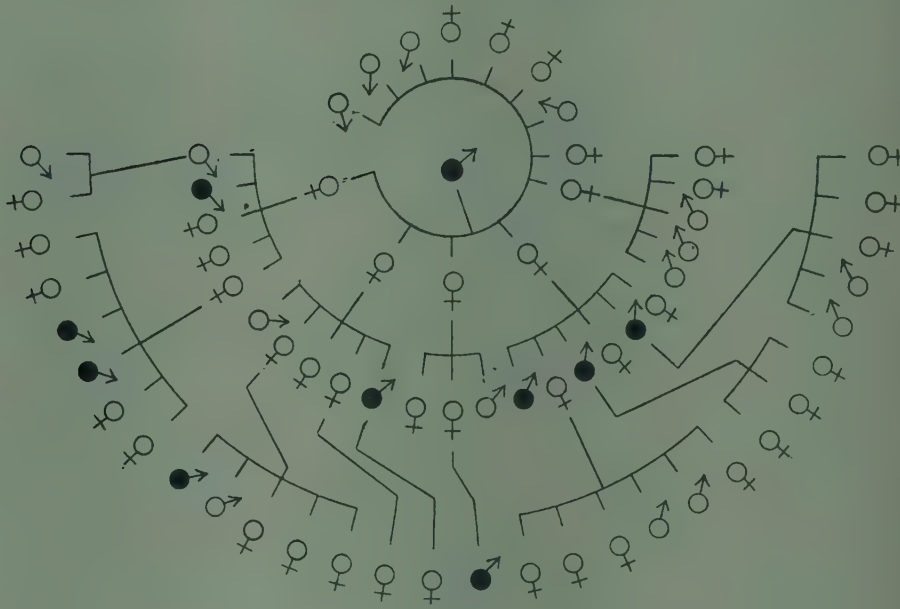


FIG. 53.
Night-blindness with myopia, after Varelmann (Extract).

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[1] Ueber den grossen schweizerischen Stammbaum, in dem mit Kurzsichtigkeit kombinierte Nachtblindheit sich forterbt, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XV, no. 1, 1923.

[2] Die Vererbung der Hemeralopie mit Myopie, "Archiv für Augenheilkunde", vol. XCVI, nos. 3 and 4, 1925.

[3] Ueber eine bis jetzt nicht bekannte Verknüpfung hochgradiger Myopie mit angeborener Hermeralopie, "Archiv der Julius-Klaus-Stiftung", vol. I, nos. 3 and 4, 1925.

[4] Kawakami, Ueber die Vererbung der Oguchischen Krankheit, "Klinische Monatsblätter für Augenheilkunde", vol. LXXII, 1924.

We may suppose that the ordinary sex-linked form of night-blindness is due to the lack of a particular heredity factor in the sex-chromosome, whereas the recessive form last described may depend upon the lack of a heredity factor in some other chromosome, and the dominant kind may depend upon the existence of an abnormal heredity factor in one of the other chromosomes—a factor which prevents the development of the faculty for seeing in a half-light. Vision in a poor light depends upon other retinal elements than vision in broad daylight. Daylight vision is a function of the cones of the retina, which are also concerned in colour-vision. Vision in "darkness", on the other hand, is a function of the rods of the retina, which are concerned in providing us with colourless images such as those we see in a moonlit landscape. The activity of the cones, the retinal elements which give us colour sensations, is dependent upon a strong light, and they are therefore functionless in the "dark".

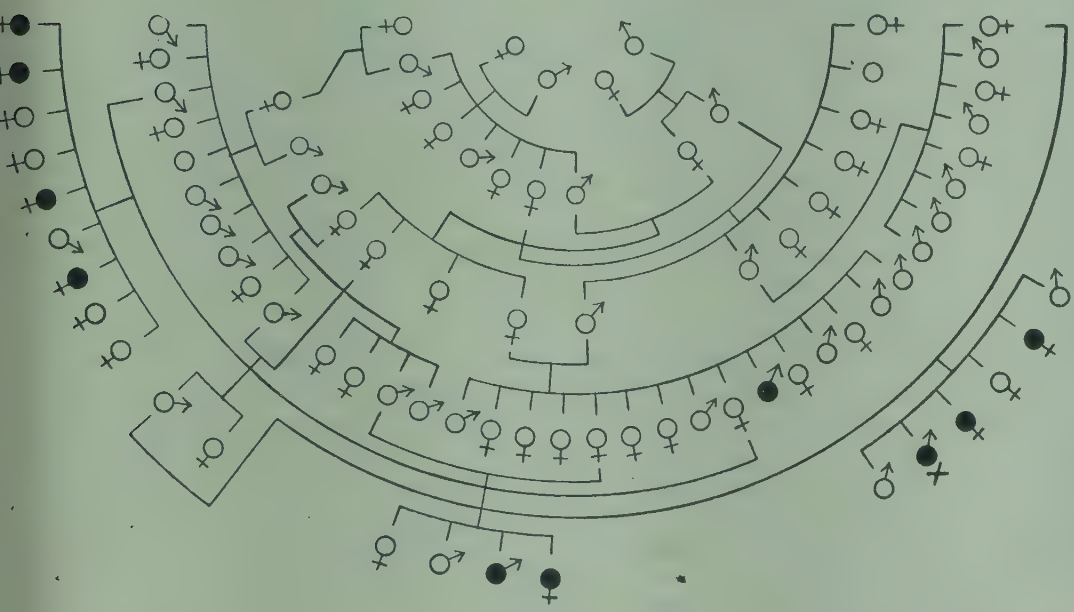

Fig. 54.

ligh-grade night-blindness with high-grade myopia, after Vogt and Gassler. The night-blind man marked in the tree with a cross was exceptional in being free from myopia.

Now, there is a hereditary malady in which (in contradistinction to what happens in night-blindness) it is the function of the retinal cones that is disordered. Persons suffering from this complaint are equipped with only that part of the sense of vision by means of which normal persons are able to see in moonlight (for instance), this being a part of the visual function which is excluded from operation in a strong light. In England, therefore, persons thus affected are said to suffer from *day-blindness*, but here in Germany we call it (less appropriately) "total colour-blindness", because the impairment of the function of the cones makes the sufferers colour-blind as well as day-blind. The malady is recessive.

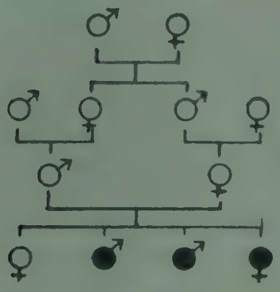

FIG. 55.
Day-blindness, after R. Hessberg.[1]

The tree reproduced in Fig. 55 shows us how from a marriage between first-cousins (the sort of marriage which is so apt to result in the coincidence of identical recessive taints) there sprang three day-blind children. It is, of course, exceptional to find three affected persons among four children, for in recessive maladies and when both parents are heterozygotic each child's chance of being affected is only 1 : 4. We must not be surprised, therefore, to find that in many families among a great number of brothers and sisters only one may exhibit a recessive hereditary malady, as we see, for instance, in the following tree (Fig 56).

Those without special experience in the heredity of day-blindness and devoid of expert knowledge of genetics will hardly be inclined to suppose that an isolated case of day-blindness such as is figured

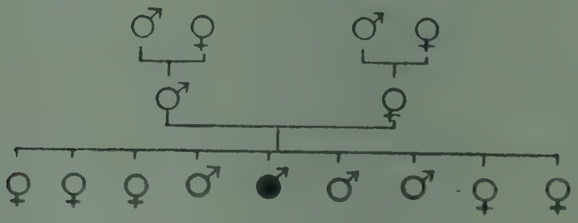

FIG. 56.

Day-blindness, after A. Lutz.[2]

above can have been of hereditary causation. When only one among eight brothers and sisters has a trouble of the kind, and when the parents and the grandparents are all healthy, many doctors would conclude, even to-day, that heredity could have nothing to do with the matter.

[1] Ein Beitrag zur angeborenen totalen Farbenblindheit, "Klinische Monatsblätter für Augenheilkunde", vol. XLVII, no. 2, 1909.

[2] Ueber einige Stammbäume und die Anwendung der mendelschen Regeln auf die Ophthalmologie, "Archiv für Ophthalmologie", vol. LXXIX, no. 3, 1911.

A fairly extensive study has been made concerning the heredity of *partial colour-blindness* or *red-green blindness.*

The colour-perceiving cone apparatus of the eye, which in day-blindness is completely functionless, has in certain other hereditary disorders lost only part of its function. The elements of or the substances in the retina upon which the perception of yellow and blue depends are different from those on which the perception of red and green depends. Marked disturbances of the colour-vision for yellow and blue are extremely rare, but those affecting the colour-vision for red and green are common. Statistical data show that from 3 to 4 % of all males are red-green blind, whereas the proportion of red-green blind persons in the female sex appears to be only about one tenth of that. At least as common, however, are slight anomalies of the red-green sense. J. Schiötz,[1] examining 2005 boys, found that 215 exhibited disturbances of the red-green sense (= 10·7 ± 0·67 %); and examining 2200 girls found only 20 thus affected (= 0·91 ± 0·21 %).

As a rule, however, persons regarded as red-green blind are not wholly incapable of distinguishing between red and green light. There are many circumstances in which, for practical purposes, such persons are colour-blind, so that, for instance, they are unfitted to become railwaymen. Nevertheless they retain a vestige of the red-green sense, and this enables them, in favourable circumstances, to distinguish red from green when these colours have a high degree of saturation, and when the red and green areas are large. Absolute red-green blindness is certainly very rare, if it ever occurs at all. The methods used by ophthalmologists to detect colour-blindness are of practical value in showing the existence of more or less weakness of the red-green sense, but they are not calculated to disclose the retention of vestiges of that sense.

A normal-sighted person can study the varying degrees of his own colour-vision by testing it in twilight. When the light is not too faint, he will in twilight see colours much as the red-green blind person sees them in broad daylight. As the light fails, the first element in colour perception to disappear is the power of distinguishing red from green, at a time when the power of distinguishing blue from yellow is still fully preserved. Then, with a yet further diminution in the intensity of the light, the normal-sighted person loses also the power of distinguishing blue from yellow. In moonlight we are all colour-blind.

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[1] Rotgrünblindheit als Erbeigenschaft, "Klinische Monatsblätter für Augenheilkunde", vol. LXVIII, 1922.

F. Horner,[1] in the year 1876, described a family in which red-green blindness could be traced for eight generations. Among the males, fifteen suffered from this affection, whereas all the women had normal colour-vision. The heredity was recessive sex-linked, and the same type of heredity has since been noted in many other genealogical trees showing red-green blindness. Since Horner failed to include the normal-sighted males in his genealogical tree, it does not give a satisfactory picture of the heredity of red-green blindness, and I therefore reproduce in its stead a family tree published by A. Groenouw.[2]

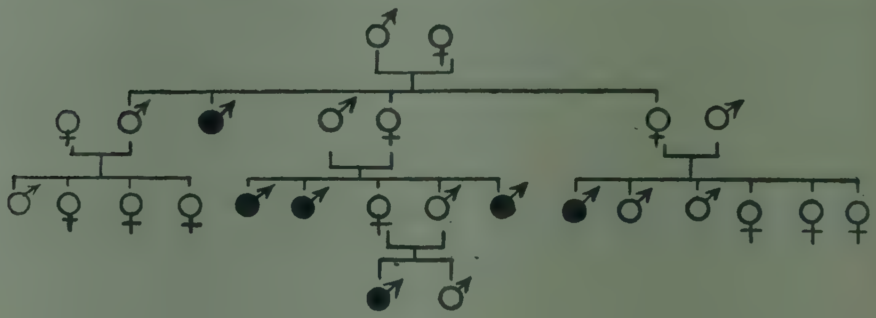

FIG. 57.

Red-green blindness, after Groenouw.

In contradistinction to most of the anomalies whose heredity is recessive sex-linked, red-green blindness is not infrequently met with in females. A family tree illustrating this has been published by the physiologist W. A. Nagel.[3]

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[1] Die Erblichkeit des Daltonismus, ein Beitrag zum Vererbungsgesetz. Amtlicher Bericht über die Verwaltung des Medizinalwesens des Kantons Zürichs vom Jahre 1876.

[2] Die Beziehungen der Allgemeinleiden und Organerkrankungen zu Veränderungen und Krankheiten des Sehorgans, in Graefe and Saemisch's Handbuch der gesamten Augenheilkunde, third edition, 1920.

[3] Eine Dichromatenfamilie, "Zeitschr. für Pathologie und Physiologie der Sinnesorgane", vol. XLI, 1907.

On the theory of sex-linked heredity, we must assume, in the case of the family whose genealogical tree is shown in Fig. 58, that the wife of the progenitor suffering from red-green blindness was herself likewise affected with the taint, though in a masked form. In that case all the female children of the pair would have had a 50 % chance of becoming red-green blind. The sons of a woman who is herself red-green blind must themselves all suffer from the malady. The tree will be seen to conform with both these suppositions.

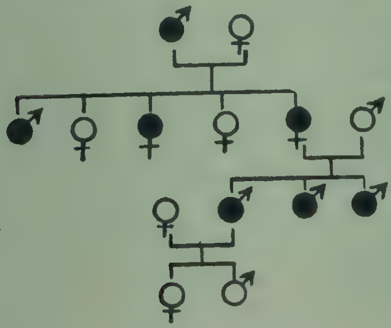

FIG. 58.

A family in which some of the females are red-green blind as well as some of the males. After Nagel.

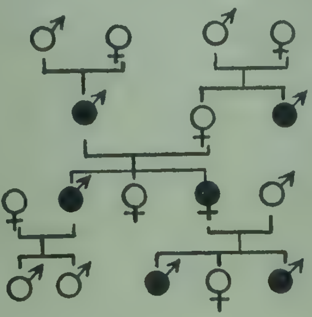

FIG. 59.

A family in which one of the females is red-green blind as well as five of the males. After Lort.

No less interesting is the family tree which was published by M. Lort[1] as long ago as the year 1778 (Fig. 59).

In order to test the inferences drawn from the theory of sex-linked heredity, the ophthalmologist A. Vogt[2] has made a systematic search for families in which there were red-green blind daughters, and has been able to draw up several genealogical trees which admirably confirm the theory. He also came across a family in which the two parents and all three of their children (two daughters and one son) were red-green blind—this inheritance being exactly what the theory would have led us to expect. Another family of the same kind was described by G. Göthlin.[3] In that case,

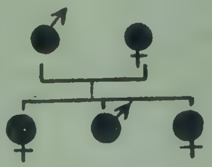

FIG. 60.

Red-green blindness, after Vogt.

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[1] The Family Scott, "Philosophical Transactions", vol. XVIII, 1778.

[2] Ueber geschlechtsgebundene Vererbung von Augenleiden, "Schweiz. Medizin. Wochenschr.", no. 4, 1921.

[3] Congenital Red-Green Abnormality in Colour-Vision, etc. "Acta Ophthalmologien", Copenhagen, 1924.

both parents were green blind and so were all their six children (three sons and three daughters).

We distinguish between two groups of disturbances of the red-green colour sense: *red blindness*, or *protanopia*; and *green blindness*, or *deuteranopia*. In both, the power of perceiving red and the power of perceiving green are alike reduced; but to the red-blind persons, red objects appear darker than green ones, whereas to the green-blind persons, green objects appear darker than red ones. F. Holmgren[1] long ago pointed out that these two kinds of weakness of the red-green colour sense are separately inheritable. The fact has been recently confirmed by E. Wölfflin[2] and G. Göthlin, and substantiated by genealogical trees. We do not know as yet what happens when the heredity factor producing red-blindness and the heredity factor producing green-blindness come together in the same person.

The heredity factors respectively producing green-blindness and the somewhat rarer red-blindness being specifically distinct, we find that the degree of disturbance of the colour sense produced by either factor in a particular family may vary considerably; we do not find that one and the same heredity factor, acting with more or with less intensity, will produce in one member of a family weakness of the green sense and in another member weakness of the red sense, but what happens is (for instance) that the factor which produces weakness of the green sense will in one member of the family produce that weakness to a high degree, and in another member to a low degree. Wölfflin has been able to substantiate this also. Thus the heredity of general weakness of the colour sense or of the anomalous trichromasy is on all fours with that of partial colour blindness or dichromasy.

One possible supposition is that in the case of the heredity factors determining respectively red-blindness and green-blindness we have to do with multiple allelomorphy. It is also conceivable that different degrees of disturbance of the red-green sense are determined by different multiple allelomorphs; but we have no certain knowledge of this matter. G. Just,[3] basing his argument on a genealogical tree published by Doederlein,[4] has suggested that exceptional (hypernormal) sensibility to red (sometimes called "red-sight") may be caused by a heredity factor which stands in an allelomorphic relation to the factor causing red-green blindness, but I do not consider that Doederlein's family tree is enough to substantiate such a theory. We must also remember that sex-linked heredity factors do not necessarily all stand in an allelomorphic relation one to another, seeing that the sex chromosomes presumably contain many genes.

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[1] Ueber die Farbenblindheit in Schweden, "Zentralblatt für Augenheilkunde", 1878.

[2] Ueber das Vererbungsgesetz der anomalen Trichromaten, "Pflügers Archiv für die gesamte Physiologie", vol. CCI, nos. 1 and 2, 1923.

[3] Zur Vererbung der Farbensinnstufen beim Menschen, "Archiv für Augenheilkunde", vol. XCVI, nos. 3 and 4, 1925.

[4] Ueber die Vererbung von Farbensinnstörungen, "Archiv für Augenheilkunde", vol. XC, 1921.

H. W. Siemens[1] has reported a case in which a manifestly heterozygotic woman was green-blind, whereas hitherto all heterozygotic women have been found to have the colour sense intact.

Tumours of the retina (gliomata) will be considered in the subsection on Cancer and other Malignant Tumours.

3. HEREDITARY DISEASES OF THE EAR

The heredity of *deaf-mutism* has long aroused the interest of investigators. A. G. Bell,[2] the inventor of the telephone, wrote a paper in the year 1884 upon deaf-mutism as a family peculiarity. At this early date, too, he recognised that the heredity factors giving rise to deaf-mutism are not essentially different from other racial characters. Building on the foundations laid by Bell, the philologist E. A. Fay[3] collected an enormous amount of material relating to 4471 marriages entered into by deaf-mutes, the bulk of the data being secured by correspondence. Although in the circumstances most of this material was far from trustworthy, by a critical study of the family trees published by Bell and Fay, the Swedish racial biologist H. Lundborg[4] was able to show with a high degree of probability that the heredity of deaf-mutism is simply recessive. In the year 1923 W. Albrecht,[5] professor of otology at Tübingen, published fifteen genealogical trees compiled from materials collected with all the skill of an expert, and these have enabled us to decide once for all that the heredity of deaf-mutism is simply recessive.

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[1] Ueber Manifestationsstörung bei rezessivgeschlechtsgebundener Farbenblindheit, "Sitzungsberichte der Ges. für Morphologie und Physiologie in München", vol. XXXVII, 1926.

[2] Upon the Formation of a Deaf Variety of the Human Race, "Memoirs of the National Academy of Sciences", Washington, 1883.

[3] Marriages of the Deaf in America, Washington, 1898.

[4] Ueber die Erblichkeitsverhältnisse der konstitutionellen (hereditären) Taubstummheit, "Archiv für Rassen- und Gesellschaftsbiologie", vol. IX, no. 2, 1912.

[5] Ueber die Vererbung der konstitutionell-sporadischen Taubstummheit, der hereditären Labyrinthschwerhörigkeit und der Otosklerose, "Archiv für Ohrenheilkunde", vol. CX, no. 1, 1923.

It must be noted, however, that morbid heredity has not been the determining factor in all cases of deaf-mutism. That disorder may also arise as a sequel of certain infectious diseases, such as meningitis and scarlet fever, by which in early childhood the auditory mechanism of the internal ear may be destroyed. Furthermore, congenital deaf-mutism is not infrequently an outcome of syphilis. In regions where (from causes which have not yet been fully elucidated—perhaps a lack of iodine) cretinism and goitre prevail, deaf-mutism is also fairly common in association with cretinism.

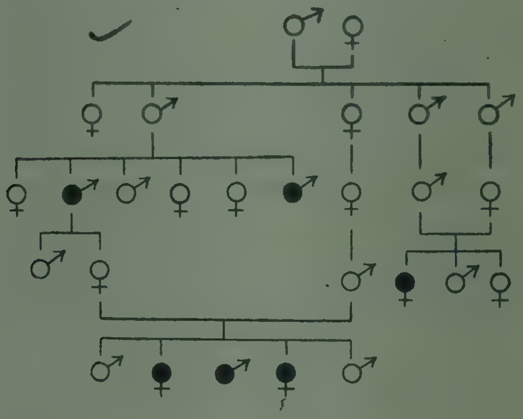

Fig. 61.

Deaf-mutism, after Albrecht. Infants dying in the first year of life have been omitted from the tree.

In Germany, there are about 50,000 deaf-mutes, and it is estimated that from one fourth to one third of these owe their infirmity to morbid heredity.

Fig. 61 shows a family tree recorded by Albrecht, demonstrating the recessive heredity of deaf-mutism. Two of the three sets of brothers and sisters among whom there were deaf-mutes had sprung from consanguineous marriages.

In all, in the family trees given by Albrecht, we find thirty sets of brothers and sisters each containing at least one deaf-mute. Ten of these sets (approximately 33 %) were the issue of consanguineous marriages, and five sets (approximately 17 %)

were the offspring of the marriage of first cousins. There is good reason to suppose that if in all the cases it had been possible to trace back the ancestry for a long period, the number of consanguineous marriages would have been larger. H. Mygind[1] found that 9 % of all the deaf-mutes who came under his notice and 23 % of the deaf-mutes known to have been born deaf were the offspring of consanguineous marriages; Schönlank[2] found 21 % of consanguineous marriages; V. Hammerschlag[3] found 7 % of consanguineous marriages in the parentage of all deaf-mutes and from 30 to 40 % in the parentage of those known to have been born deaf. Since in the general population the proportion of persons who are the offspring of the marriage of near kin does not amount to 1 %, the very high proportion of consanguineous marriages among the parents of deaf-mutes supplies further evidence that the heredity of this disorder is recessive.

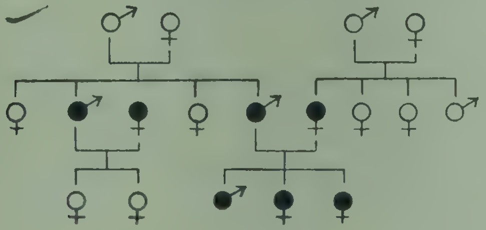

FIG. 62.
Deaf-mutism, after Albrecht.

When two persons affected with the same kind of hereditary deaf-mutism marry one another, the theory of a recessive heredity would lead us to expect that all the offspring of such a marriage would be deaf-mutes. Among Albrecht's family trees we find one such instance, which is reproduced in Fig. 62.

Here all three children sprung from the marriage of two deaf-mutes are themselves deaf-mutes. In the left side of the same figure, however, we see two normal-hearing daughters spring from the marriage of parents both of whom are deaf-mutes—the explanation being that in this case the mother was not a hereditary deaf-mute, but became totally deaf in early childhood as a sequel of scarlet fever. Acquired deaf-mutism, the outcome of an environmental cause, is, of course, not transmissible by inheritance. In Fay's material, likewise, we find families in which deaf-mutism breeds absolutely true. Two such genealogical trees are reproduced in Figs. 63 and 64. Fig. 63 shows a family in which both parents were deaf-mute but all their four children had normal hearing. Here the deaf-mutism of the father was acquired, having been a sequel of some febrile disorder.

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[1] Die angeborene Taubheit, Hirschwald, Berlin, 1890.

[2] Ergebnisse einer zweiten Untersuchungsreihe von Taubstummen in Zürich, "Schweizer. Rundschau für Medizin", 1920.

[3] Ueber die Beziehungen zwischen hereditärdegenerativen Taubstummheit und der Konsanguinität der Erzeuger, "Zeitschrift für Ohrenheilkunde", vol. XLVII, 1904.

H. Orth[1] has reported concerning two families in which both the parents and all the children (five in each case) were deaf-mutes.

Connected with the recessive heredity of deaf-mutism is the circumstance that (as E. Hanhart,[2] among others, has pointed out) it is comparatively common in regions where inbreeding is rife (isolated valleys, for instance). Before Hanhart, Albrecht had noted that in Wurtemberg deaf-mutism was especially prevalent in regions where consanguineous marriages were exceptionally common—as, for instance, in Catholic enclaves surrounded by a Protestant population.

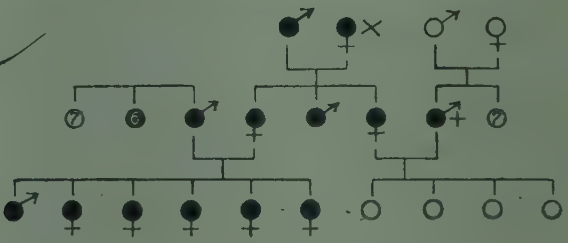

FIG. 63.

Deaf-mutism, after Fay.

× = deaf-mutism probably a sequel of measles in the second year of life.
+ = deaf-mutism a sequel of a febrile disorder in the second year of life.

E. Finkbeiner,[3] after a study of the Swiss census of 1870, was able to show that in places having a population of less than 100 persons there were 9·2 deaf-mutes per thousand; in places having a population ranging from 100 to 200, 5·7 per thousand; in places with a population ranging from 200 to 300, 4·5 per thousand; and in places with a population above 300, 3·4 per thousand.

Owing to the greater frequency of consanguineous marriages among Jews, deaf-mutism is commoner in the Jewish than in the Gentile population. In Berlin, there are 27 deaf-mutes per 10,000 Jews, as against 6 deaf-mutes per 10,000 Gentiles. Since we have no warrant for assuming that deaf-mutism due to external causes (scarlet fever, etc.) is to that extent commoner among Jews, we can only suppose that the comparative frequency of deaf-mutism in Jews must be mainly due to hereditary causes. Seeing that the much greater frequency of consanguineous marriages among Jews supplies a satisfactory explanation, there is no reason for invoking a peculiar "racial predisposition". |)

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[1] Zum Erbgang der konstitutionellen Taubstummheit, "Archiv für Ohren-, Nasen- und Kehlkopfheilkunde", vol. CXI, 1923, p. 84.

[2] Ueber die Bedeutung der Erforschung von Inzuchtsgebieten, "Schweizerische Medizinische Wochenschrift", no. 50, 1924.

[3] Die kretinischer Entartung, Springer, Berlin, 1923.

(A notable proportion of all deaf-mutes are simultaneously feeble-minded; approximately 30 % as against approximately 1·5 % in the general population. Blindness is also several times as common among deaf-mutes and among the brothers and sisters of deaf-mutes as in the general population, occurring especially in the form of retinal atrophy (retinitis pigmentosa). Albrecht has expressed the opinion that this coincidence of the different taints is the outcome of consanguineous marriages, thanks to which not only the disposition to deaf-mutism, but also the disposition to other recessive hereditary maladies has become manifest.) Certainly this explanation may be accepted as accounting for the majority of the instances. Still, we must remember that the external causes which are capable of producing deaf-mutism (syphilis, meningitis), may have left other injuries. As to whether there are certain heredity factors which can produce other troubles in conjunction with deaf-mutism, we have as yet no information.

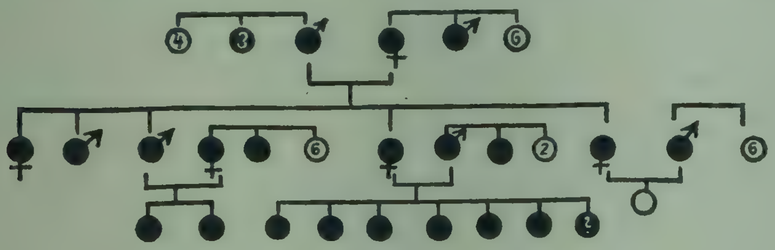

FIG. 64.

Deaf-mutism, after Fay. As regards the case in the last row, marked with a query, there is no certain information as to whether the person was deaf.

As regards the pathological anatomy of hereditary deaf-mutism, the affection is dependent upon a congenital lack of the nerve elements of the internal ear, and apparently also of certain parts of the brain which are concerned in the perception of sound. But, as the outcome of hereditary causation, the nerve elements of the internal ear may perish in middle life and be replaced by connective tissue. This is the anatomical foundation of *hereditary labyrinthine deafness*, or *internal-ear deafness*. We can also speak of *auditory-nerve atrophy*. The trouble usually begins to be noted at about the age of forty, the loss of hearing being a slow process which may take several years to complete but is sometimes more rapid in its progress. It may go on until absolute deafness ensues. Albrecht has published ten family trees and these indicate a dominant heredity.

The auditory nerve can also be injured by external influences, such as syphilis, scarlet fever, and influenza; arteriosclerosis; such poisons as quinine; persistent exposure to loud noises. As regards the last-named influence, since only in exceptional instances do boilermakers or artillerists become deaf, we must presume that there is a hereditary factor likewise at work. The so-called *senile deafness* also appears to run in families, and to be in great measure the outcome of a hereditary predisposition. Probably we have to do with a labyrinthine deafness of late onset. Since it is the exception rather than the rule for old people to become deaf, we cannot regard advancing years as in themselves the efficient cause.

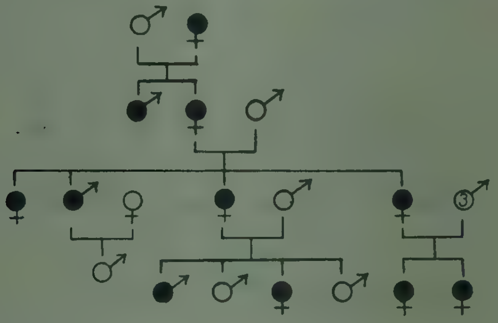

FIG. 65.
Deafness (auditory-nerve atrophy), after Albrecht.

Quite distinct from auditory-nerve atrophy is another form of progressive deafness which is known as *otosclerosis*. This commonly begins at about the thirtieth year of life. The decline in the faculty of hearing is slow but continuous, until in the end the patient can hear very little; but absolute deafness does not generally ensue in otosclerosis. This form of deafness is almost always attended by subjective "noises in the ears", which are a very troublesome accompaniment. Anatomically, we find changes in the bony walls of the internal ear. According to Albrecht, there is no biological unity corresponding to the clinical picture of otosclerosis. In some families, a dominant heredity has been observed. One such genealogical tree is shown in Fig. 66. When, as sometimes happens, the malady seems to skip a generation, we may suppose that the changes in the bones may have none the less occurred, but not in a region where they induce defect of hearing. Otosclerosis also occurs as an accompaniment of osteopsathyrosis (see below); in genealogical trees illustrating the occurrence of this combination, the dominance of otosclerosis is still more irregular. For the rest, not all cases of otosclerosis depend upon dominant heredity factors. Only in from one third to one half of the cases have otosclerotics relatives suffering from deafness. Perhaps there may also be recessive heredity factors giving rise to otosclerosis, but this hypothesis has not been substantiated.

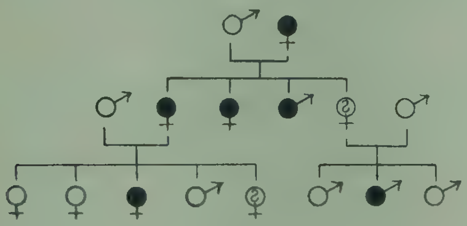

FIG. 66.
Deafness (otosclerosis), after Albrecht.

J. Bauer and C. Stein[1] have advocated the theory that deaf-mutism, labyrinthine deafness, and otosclerosis are caused by the same heredity factors; but their paper is invalidated by a lack of sound methodology. It has been amply proved that these three maladies are biologically distinct.

Another important cause of deafness is suppurative inflammation of the middle ear (*otitis media*), which in a certain proportion of the cases leads to destruction of the sound-conduction apparatus in the middle ear, and thus causes deafness. This affection manifestly tends to run in families, though it appears as the outcome of the most various infections, such as measles, scarlet fever, tonsillitis, or a simple cold in the head. C. Stein[2] has described a number of such families, in which a hereditary predisposition to the onset of deafness is obviously of more moment than the infection to which the predisposed persons fall a prey sooner or later. There seem to be affinities between this predisposition and the lymphatic or adenoid diathesis (see below, p. 329).

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[1] Vererbung und Konstitution bei Ohrenkrankheiten, "Zeitschrift für Konstitutionslehre", vol. X, no. 5, 1925.

[2] Gehörorgan und Konstitution, "Zeitschr. für Ohrenheilkunde", vol. LXXVI, 1917.

W. Weitz, in his study of twins,[1] found three pairs of mon-oval twins in which both members of the pair suffered from suppurative inflammation of the middle ear. Two twin sisters had in early childhood simultaneously suffered from "running ears", and at the age of eight and a half years both of them suffered for the second time from a suppurative inflammation of the middle ear, on this occasion as a sequel of scarlet fever. J. Paulsen has published similar observations on two pairs of twins.[2]

4. HEREDITARY DISEASES OF THE SKIN

Of late years, our knowledge of the heredity of skin diseases has been greatly augmented by H. W. Siemens.[3] The form of *partial albinism* in which *white patches* appear on the skin, and which is common in all kinds of domesticated animals (especially in cattle), is not very rare in man. Such albinotic patches are especially conspicuous in members of dark-skinned races (in Germany we call them "Elsterneger"—"magpie niggers" or "piebald niggers"). In domesticated animals this character of being flecked with white is dominant, and in the East Frisian race of cattle it is deliberately bred as a racial character. In human beings, likewise, such flecking with white is generally dominant. A good many genealogical trees showing its family incidence have been published.[4] In different families, this tendency to be "piebald" is developed to a very varying degree, one of the slightest forms being that which leads to the appearance of a white forelock. Although in any particular family those affected show a rough correspondence in the general extent and the distribution of the white patches, there is a certain amount of individual variation in respect of the size, shape, and situation of the spots. Thus, the same heredity factor at work in two different members of a family may produce in one a splash of white skin on the hairless forehead, and in another a tuft of white hair on the top of the head.

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[1] Studien an eineiigen Zwillingen, "Zeitschr. für klinische Medizin", vol. CI, nos. 1 and 2, 1924.

[2] Beobachtungen an eineiigen Zwillingen, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XVII, no. 2, 1925.

[3] Die spezielle Vererbungspathologie der Haut, "Virchow's Archiv", vol. CCXXXVIII, 1922.—Einführung in die allgemeine und spezielle Vererbungspathologie, second edition, Springer, Berlin, 1923.

[4] K. Pearson, E. Nettleship, C. H. Usher, Albinism in Man, three vols., Dulau, London, 1911.

The probable way in which these white patches are formed is as follows. It seems that an enzyme must collaborate in the formation of the pigment in the skin and the hair, and that normally this enzyme is produced at an early stage of development. When the enzyme is lacking in certain areas, they remain devoid of pigment. An analogous process occurs in the tinting of horse-chestnuts. As every one knows, these are white to begin with; then sharply circumscribed shining reddish-brown spots appear on the surface, spreading at the edges, until ultimately the whole surface of the horse-chestnut becomes brown. If, owing to an inadequacy in the supply of enzyme, this process of browning could not be completed, white patches would remain here and there. The white flecking in domesticated animals and human beings seems to arise in some such way. When, owing to a weakness in particular heredity factors, the enzyme needed for the extension of pigment all over the skin is formed in inadequate quantities, unpigmented patches remain. This explains why the white flecking is usually asymmetrical, although the asymmetry is in most instances of a moderate degree.

In man, such white patches are sometimes technically termed *naevi depigmentosi*. E. Meirowsky[1] regards the formation of naevi in general as analogous to asymmetrical flecking of the skin. A *naevus*, or *birthmark*, is an "island" of abnormal elements in the skin. As a rule such "islands" are more darkly pigmented than the rest of the skin, and are often exceptionally hairy. Small, very slightly raised brown naevi (in common parlance known as moles) are so frequent that almost every adult human being has some of them. As a rule they are not present at birth, and only develop during the period of growth. H. W. Siemens,[2] studying the resemblance between mon-oval twins as regards the number of their naevi and examining 45 pairs of twins for this purpose, has discovered a correlation-coefficient (see below, pp. 541 et seq.) of 0·8, whereas in 23 pairs of di-oval twins the correlation-coefficient was 0·4. Meirowsky[1] has ascertained the figures relating to the naevi in 300 pairs of twins. From his results, excluding the disturbing influence of age, I have deduced for mon-oval twins a correlation-coefficient of 0·78, and for di-oval twins one of 0·31. Such correlations and the contrast between them are what we should expect to find if the importance of heredity in the production of pigmentary naevi were several times as great as the importance of environmental influences in their production.

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[1] Ueber die Entstehung der sogenannten kongenitalen Missbildungen der Haut, Vienna and Leipzig, 1919.

[2] Ueber die Bedeutung der Erbanlagen für die Entstehung der Muttermäler, "Archiv für Dermatologie", vol. CXLVII, no. 1, 1924.

When people speak of a "birthmark" without qualification, just as when medical practitioners speak of a "naevus" without qualification, they generally mean one of the raised islands of unduly vascular tissue, reddish in colour, which when large and comparatively flat (especially on the face) are known as "*port-wine marks*". Technically, they are also known as *angiomata.* Heredity is especially determinative in the production of a naevus localised on the nape of the neck, the so-called *naevus of Unna*; in the case of other naevi, the influence of heredity is less conspicuous. Such birthmarks are composed of masses of vascular tissue, which sometimes disfigure the skin throughout an area as large as a small plate.

The much smaller localised dilatation of capillaries and minute arteries in the skin, known technically as *telangiectases*, are, according to Siemens and Henle,[2] dominant anomalies.

A naevoid affection originating in the cutaneous nerves forms what are known as *neurofibromata* (*Recklinghausen's disease*). Concerning this malady, S. A. Preiser and C. B. Davenport[3] have published some family trees showing a dominant heredity with interruptions (irregular dominance).

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[1] Zwillingsbiologische Untersuchungen, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XVIII, no. 3, 1926.

[2] K. Henle, Gefässmäler und Teleangiectasien, "Archiv für Dermatologie", vol. CXLIII, no. 3, 1923.

[3] Multiple Neuro-fibromatosis, etc., "Eugenics Record Office Bulletin", no. 19, 1910.

We learn from H. W. Siemens,[1] however, that the tendency of Recklinghausen's disease to run in families is not marked. It would seem that in some cases the underlying heredity factor causes nothing more than abnormal areas of pigmentation. In a notable proportion of cases, persons affected with neuro-fibromatosis succumb in the end to malignant tumours which develop out of the naevi or fibromata.

In cases of *tuberous sclerosis* there develops, as a rule, in addition to little tuberous growths in the central nervous system, naevus-like growths on the face and neck (*adenoma sebaceum*), and often growths in the kidneys and in the heart. The same heredity factor acting on various members of one family, may lead to the growth of such little tumours, now in one of these organs, and now in another.[2]

According to Hammer[3] and Meirowsky, a marked development of *freckles* (*ephelides*) is generally the outcome of dominant heredity factors. Mon-oval twins are extraordinarily alike in this matter of freckling. H. W. Siemens'[4] observations on di-oval twins indicate that several heredity factors cooperate in producing freckles. There is a correlation with red pigmentation of the hair. The tendency to the formation of freckles seems to be epistatically masked by dark pigmentation.

Xanthomatosis, characterised by the formation of dark-yellow somewhat raised patches on the skin, is the outcome of the presence of an excessive quantity of cholesterin in the blood, with a consequent deposit of cholesterin in the skin. The heredity of this malady is in most cases dominant, but the taint does not manifest itself in all persons who carry it.[5]

An extremely malignant disorder is the one known as *xeroderma pigmentosum*. Children suffering from this malady are born with a skin apparently normal, but (as an effect of sunlight, and usually during the first years of life) inflammatory areas make their appearance on the exposed parts of the skin—the face and the hands. In these areas there is a freckle-like pigmentation attended by contraction similar to that which occurs in scar-tissue, followed sooner or later in most cases by cancerous changes in the affected areas so that persons with xeroderma pigmentosum usually die in the first or second decade, and only in very rare instances attain the age of forty. H. W. Siemens and E. Kohn[1] were able to collect the records of 333 cases, belonging to 222 families. The parents of the patients had never suffered from the disease—and this immunity was to be expected, seeing that so few sufferers from xeroderma pigmentosum attain the age of reproduction. Studying the ratio of the incidence of the malady among brothers and sisters, Siemens found approximately 1 patient to 3·4 healthy persons, a result in which the margin of error inevitable in dealing with so small a number of cases does not exceed what is to be expected when compared with the mendelian expectation of a ratio of 1 : 3 for recessive heredity. In 25 % of the instances, the parents were blood relations; and in 20 %, they were first cousins. This confirms the view that the heredity is simply recessive.

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[1] Aetiologisch-dermatologische Studien über die Recklinghausensche Krankheit, "Virchow's Archiv", vol. CCLX, no. 1, 1926.

[2] H. W. Siemens, op. cit.

[3] Ueber die mendelsche Vererbung beim Menschen, "Medizinische Klinik", 1912.

[4] Die Zwillingspathologie, Berlin, 1924.

[5] A. Fasold, Xanthom (Siemens' Studien über Vererbung von Hautkrankheiten, VI) "Archiv für Rassenbiologie", vol. XVI, no. 1, 1924.

Velhagen[2] has reported on a family circle in which three brothers married three sisters. From two of these marriages, children with xeroderma pigmentosum were born: in one of the families three of the children out of seven being affected; and in the other family there being only two children, both suffered from the disease. Among the Jewish population, xeroderma is commoner than among Gentiles, and in Siemens' view this is probably the outcome of the greater frequency of consanguineous marriages among Jews.

A far less serious but still undue sensitiveness of the skin to sunlight is seen in the rare disease known as *hydroa aestivale*, in which, as an effect of strong exposure to short light-rays, and sometimes even in winter owing to the reflection of sunlight from snow, blebs form on the skin, and may leave scars when they have healed. Hitherto this disease has been observed chiefly in males. According to Siemens, it may be supposed to depend upon a recessive heredity, which, however, does not appear to be sex-linked.

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[1] Xeroderma pigmentosum (Studien über Vererbung von Hautkrankheiten, IX), "Zeitschrift für induktive Abstammungs- und Vererbungslehre", vol. XXXVIII, 1925, p. 1.

[2] Beitrag zur Kenntnis des Xeroderma pigmentosum, "Archiv für Augenheilkunde", 1903.

Susceptibility to cold, like susceptibility to sunlight seems to be hereditary. Certain persons only are liable to *chilblains*; and in those not liable, exposure to cold does not produce them. A disease which certainly does run in families is *symmetrical gangrene* or *Raynaud's disease*, in which, presumably owing to a disturbance of the internal secretions (perhaps of the pituitary), there occurs a vascular spasm in the extremities, leading when severe to gangrene of the fingers or toes—the same on both sides. Grote had a patient suffering from this affection whose brother, mother, and maternal aunt had all suffered from a severe form of the disease.

Another malady due to a morbid affection of the vasomotor nerves is *hereditary chronic oedema* of the legs (known also as *hereditary trophoedema* and as *elephantiasis Arabum*), concerning which some fairly extensive family trees showing dominant heredity have been published.[1] To the accompaniment of inflammatory phenomena in the vasomotor nerves, there ensues a slowly progressive swelling of the legs, which may ultimately become so extreme that the patient is unable to walk.

Erythema exsudativum multiforme shows itself as irregularly recurrent attacks in which inflammatory red patches appear on the skin, especially on the extensor aspect of the arms and legs. In the more severe cases, blebs form on the affected areas. We have no precise knowledge as to the heredity of this disease.

Nettlerash or urticaria and *Quincke's disease* or *oedematous urticaria*, which are to some extent related to the other skin troubles we have just been considering, will be described later in connexion with the hereditary diatheses, and the exudative diathesis will be dealt with in the same connexion.

Epidermolysis bullosa traumatica (by Siemens more aptly termed *bullosis traumatica*) depends upon a state of the skin in which very slight stimuli, such as pressure, contusion, and especially friction, will cause the formation of blebs or blisters. The ordinary forms of epidermolysis have a dominant heredity. Fig. 67 shows a family tree. In one family, the incidence of the disease could be traced for seven successive generations, but in exceptional instances a generation may be skipped. The carriers of the taint in whom it does not become manifest are usually females, but there is no evidence that the malady is sex-linked.

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[1] W. Bulloch, Chronic Hereditary Triphoedema, The Treasury of Human Inheritance, Parts 1 and 2, Cambridge University Press, London, 1909.

As H. W. Siemens[1] has shown, we must distinguish from the before-mentioned ordinary form of epidermolysis in which the heredity is dominant, another form in which the heredity is recessive. The latter is known as *epidermolysis dystrophica.* This is attended by scarring and atrophy of the nails. The accompanying figure, Fig. 68, shows a family tree illustrating the heredity of this disease.

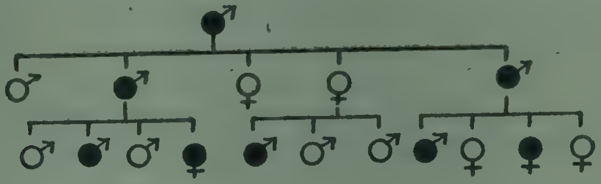

FIG. 67.

Formation of blebs or blisters on slight pressure (epidermolysis bullosa) after Colombini.

At the 1925 congress of the Schweizerische Naturforschende Gesellschaft, E. Jenny reported upon a case of *congenital bullosis.* Four children belonging to allied families where consanguineous marriages had occurred were affected with the disease to which Jenny gave the name of *epidermolysis bullosa.* I have to thank Dr. Hanhart of Zurich for drawing my attention to this family tree, part of which I reproduce in Fig. 69. Obviously the heredity is recessive. All the four children succumbed to the malady soon after birth. In a family upon which Mautner[2] has reported certain members were affected with what appears to have

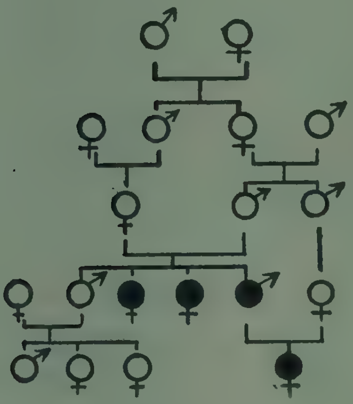

FIG. 68.

Epidermolysis bullosa dystrophica, after Sakaguchi (published by Siemens).

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[1] Literarisch-statistische Untersuchungen über die einfache und die dystrophische Form der sogenannten Epidermolysis, "Archiv für Dermatologie", vol. CXLIII, no. 3, 1923.

[2] Ueber ein familiär auftretendes letales Krankheitsbild mit Blasenbildung (Pemphigus hereditarius`. "Monasschrift für Kinderheilkunde", vol. XXII, no. 1, 1921.

been the same disease. Mautner calls it *pemphigus hereditarius*, and the name is an apt one, seeing that the blebs are not produced by any external stimulus. The malady has been frequently observed in the United States.

Mendes da Costa[1] has had under observation among blood relatives cases of a bullous disorder of the skin in which the onset was attended by severe general disturbances. The heredity was recessive and sex-linked.

The bullosis group of disorders is one in which we find the rule confirmed that the heredity of the graver morbid forms is recessive and that of the slighter morbid forms dominant. Proneness to the recessive bullosis connata (pemphigus hereditarius), in fact, represents a "lethal factor" to such an extent that if the heredity were dominant persons affected with it could not live, but would be eliminated as soon as it appeared. Simple bullosis, on the other hand, though a very troublesome complaint, does not seriously impair the individual's chances of effecting reproduction, and can therefore persist even when its heredity is dominant.

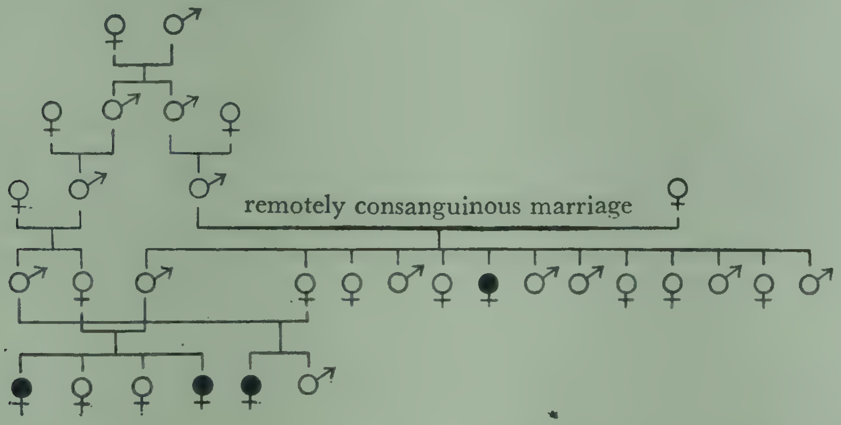

FIG. 69.
Congenital formation of blebs on the skin (Bullosis connata or pemphigus connatus), after Jenny. (Extract.)

As the outcome of heredity, the skin may manifest undue sensitiveness to chemical stimuli just as it does to mechanical stimuli. When the skin reacts to chemical stimuli by inflammatory changes attended by the outpouring of a serious secretion or by the formation of crusts and by suppuration, we speak of the condition as *eczema*. The readiness to respond to such irritant influences by eczematous inflammation varies much from individual to individual. For instance, there are some persons in whom the most fugitive contact with tar will cause eczema, whereas others can be exposed to the effect of tar for long periods without any ill-effect. There are eczematous families, wherein some of the members suffer again and again from eczema, which often arises in such persons without any obvious external cause. In what is called the constitutional eczema of infants the importance of hereditary predisposition is especially plain; for in infancy environmental influences, which in later life play so important a part in the production of eczema, have comparatively little to do with the matter (see below, the account of the exudative diathesis, p. 326).

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[1] Mendes da Costa and van der Valk, Typus maculatus der bullösen hereditären Dystrophie, "Archiv für Dermatologie", vol. XCI, no. 3, 1908.

In *Darier's disease* (*psorospermosis vegetans*), there form on the skin, and especially in the flexures of the joints and other regions where two surfaces come in contact, crusts of secretion, and often there is suppuration, and then the affected parts acquire an offensive smell. This rare condition appears to be due to a dominant heredity which is irregular in its manifestation. (Siemens and Fischer.)[1]

In *hereditary keratosis* (*keratoma*, *tylosis*), soon after birth the skin on the soles of the feet and the palms of the hands becomes horny and friable. The cornified areas are marked off from the normal skin by a bluish-red line. A considerable number of family trees showing the incidence of this malady have been published. The heredity is regularly dominant.

Hanhart has had under observation a family in which keratosis was dominant and in which the individuals affected by it suffered also from multiple fatty tumours. The keratosis made its appearance at about the age of 15 and the lipomatosis at about the age of 22. According to Siemens, it is probable that there are also recessive tendencies to keratosis.

In *hereditary ichthyosis*, the whole surface of the skin is covered with more or less dense scales or horny flakes. *Ichthyosis vulgaris* appears to be generally due to dominant heredity factors,[1] which may manifest themselves with varying severity in different members of an affected family, and in many of the carriers produce no morbid symptoms whatever. L. Leven was able in one family to trace the occurrence of ichthyosis for five generations;[2] and in another family for four generations, though here one generation was skipped.[3]

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[1] F. Fischer, Darriersche Krankheit (Siemens' Studien über Vererbung von Hautkrankheiten, VIII), "Archiv für Rassen- und Gesellschafts-biologie", vol. XVI, no. 4, 1925.

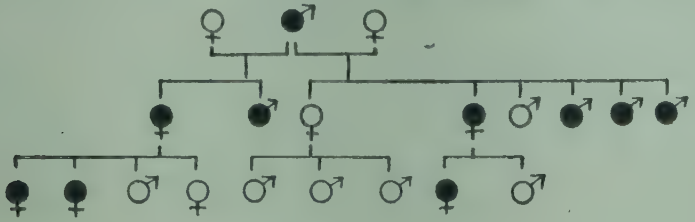

FIG. 70.
Keratosis, after Thost. (Extract only.)

Beyond question recessive in its heredity is the disease known as *ichthyosis congenita* or *keratosis universalis*, in which the skin is covered with masses of horny material. Infants thus affected are usually born prematurely and are incapable of survival. According to Siemens, 12 % of such infants are the offspring of consanguineous marriages. Very instructive

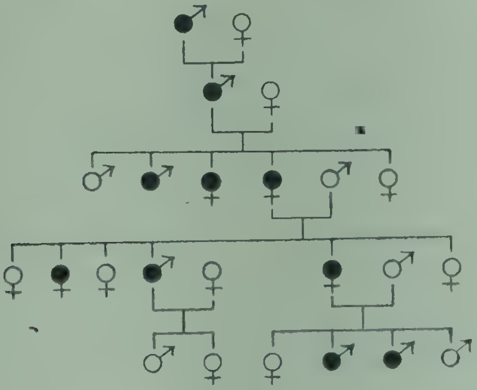

FIG. 71.
Ichthyosis vulgaris, after Leven.

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[1] E. Wehefritz, Zur Vererbung der Ichthyosis, "Zeitschrift für Konstitutionslehre", vol. X, no. 4, 1924.

[2] Stammbaum einer Ichthyosis Familie, "Archiv für Dermatologie", vol. CXXXIX, 1921, p. 117.

[3] Zur Vererbung der Lingua plicata und Ichthyosis vulgaris, "Archiv für Rassenbiologie", vol. XVI, part 3, 1925.

is the case reported by Claus in which a woman who had had five healthy children by one man, subsequently, in a union with her half-brother, had three infants affected with this disease. (See Fig. 72.)

Likewise recessive in its heredity would appear to be *erythrodermia ichthyosiformis*, regarded by Siemens as a comparatively mild form of congenital ichthyosis.

Keratosis follicularis (also known as *lichen pilaris*) is a common anomaly in which the hair follicles undergo cornification on the extensor surfaces of the arms and legs, with the result that the hairs cannot grow out of the follicles, and ultimately perish. The anomaly reaches its climax, as a rule, at about the age of puberty, when a great number of tiny tubercles on the skin in the regions named produce the picture of "goose flesh". Subsequently these horny nodules are replaced by tiny white scars. The condition is apparently caused by dominant heredity factors. Siemens found mon-oval twins suffering from the disease to be always affected to the same degree. There are transitional forms between keratosis follicularis and normal skin, and this leads us to think of polymeric causation.

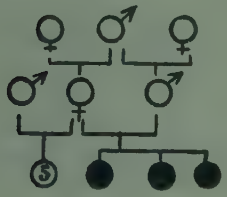

FIG. 72.
Keratosis universalis congenita, after Claus.

A peculiarly severe form of follicular keratosis has been described by H. W. Siemens[1] under the name of *keratosis follicularis spinulosa decalvans* as affecting a Bavarian family. In these patients, small spiny and horny prominences appeared on the skin, especially on that of the face and the arms. Later, in considerable areas on the nape of the neck, the vertex, and the eyebrow-regions, the prominences were replaced by scars, so that the parts thus affected became bald. In the males suffering from the disease, the eyelids were also affected, the involvement of the eyelashes in the keratosis giving rise to blepharitis (inflammation of the lids) and ultimately to opacities of the cornea. Among the female members of the family, ten had the disease in a comparatively mild form.

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[1] Ueber einen in der menschlichen Pathologie noch nicht beobachten Vererbungsmodus: dominant-geschlechtsgebundene Vererbung, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XVII, no. 1, 1925.

According to Siemens, the heredity is sex-linked, but not recessive, for if it were recessive the female carriers of the taint would be normal. On the other hand, the taint cannot be wholly dominant, seeing that the women who are affected do not suffer from the inflammation of the eyclids which forms a central feature of the disease. We might perhaps most appropriately speak of the heredity as being intermediate sex-linked. If, however, we are inclined to lay the main stress upon the blepharitis, we may speak of an incompletely recessive sex-linked heredity.

A very similar, and perhaps identical trouble, in which the heredity was likewise sex-linked, has been observed by Laméris in a Dutch family. Siemens had the opportunity of examining two male members of this family, but unfortunately did not see any of the female transmitters of the taint, and he was therefore unable to decide whether the malady was really a follicular keratosis.

Porokeratosis, or *Mibelli's disease*, is a rare malady characterised by the formation of little horny cones or raised discs growing out of the sweat glands especially on the extensor surfaces of the arms and legs. According to Siemens and E. Fulde,[1] the heredity is irregularly dominant.

Hyperidrosis, the excessive excretion of sweat, especially on the feet and hands, is apparently determined by dominant heredity factors. *Anidrosis*, a complete failure of the sweat glands to function, associated with a poor development of the teeth and the hair, was studied by Siemens in five families, and was found to be recessive sex-linked.

Psoriasis is another disease which tends to run in families.[2] This common malady shows itself by the formation of numerous circumscribed reddish-brown inflammatory areas covered with thick scales, the patches being usually restricted to the extensor surfaces of the arms and the legs. The disease is an intractable one, although persons suffering from it will often enjoy considerable periods of freedom. In severe cases, almost the whole of the body may be covered by the eruption. The mode of onset and the distribution of the disease suggest that the exciting cause must be a micro-organism. This, in conjunction with the family incidence, leads us to suppose that psoriasis is due to a hereditary susceptibility to the invasion of the skin by a still unknown parasitic fungus.

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[1] Porokeratosis Mibelli (Siemens Studien über Vererbung von Hautkrankheiten, IV), "Archiv für Dermatologie", vol. CXLIV, no. 1, 1923.

[2] S. Bettmann, Zur Aetiologie der Psoriasis, "Deutsche Medizinische Wochenschrift", 1921, p. 762.

Similar considerations apply to the causation of *lichen ruber*, a chronic disease of the skin in which groups of red papules appear upon the surface, and of which there exist a mild and a severe form. The trouble appears to be due to an infection to which the members of particular families are alone liable. According to Galewsky,[1] it seems probable that lichen ruber would more often be found to run in families if more careful attention were paid to this point.

The liability to *keloid*, this name being given to the formation of excessive quantities of scar tissue as a sequel of comparatively slight injuries to the skin, certainly tends to run in families. It is also said that negroes are especially liable to keloid.

The disease known as *seborrhoea* is a manifestation of excessive activity of the sebaceous glands. It generally runs in families, and often leads to falling-out of the hair and consequent baldness. An excessive and morbidly transformed activity of the sebaceous glands is also the cause of *acne vulgaris*, a troublesome though not dangerous disease in which, on the skin of the face, the back of the neck, and the upper parts of the skin of the trunk (both back and front), the hair follicles become plugged with sebaceous secretion, and the consequent "blackheads" have a strong tendency to become inflamed and to form pustules. The disease occurs with especial severity in adolescents (acne juvenilis), reaching its climax during the first years after the onset of puberty. Acne seems to some extent to be correlated with weakness of the constitution, nervous predisposition, and disorders of digestion. The study of twins by Siemens and Weitz has shown that mon-oval twins when affected, suffer to a similar degree from blackheads (technically termed comedones) and acne pustules. Hereditary predisposition would seem, therefore, to play an important part.

According to Weitz's observations, heredity is also of great importance in the causation of furunculosis (constitutional liability to boils). He had under observation two pairs of mon-oval twins in whom there were numerous scars of boils in the skin of the neck.

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[1] Beiträge zur Aetiologie des Lichen ruber, "Archiv für Dermatologie", vol. CXXIX, 1921, p. 484.

Loss of the hair of the head, *baldness* (*alopecia*), is apparently due to very various causes. Frequently, the loss of the hair is preceded by seborrhoea, either in the form of seborrhoea oleosa (an excessive secretion of an oily fluid by the skin), or in that of seborrhoea sicca (an excessive formation of scales by the skin of the head). In women such complete loss of hair as to deserve the name of baldness is extremely rare, but in men it is very common, and we find likewise that the more severe forms of seborrhoea occur chiefly in men. There must obviously be some connexion, here, with the hormonal activity of the testicles. It is said that eunuchs do not get bald.[1] In certain families we find that baldness tends to come on at about the same age in many or all the male members.[2] We get the general impression that the heredity is dominant. In other families we find that the men retain a good head of hair when they are well advanced in years. A falling of the hair may also result from certain poisons. Apparently the prolonged administration of mercury, as for the treatment of syphilis, favours the falling-out of the hair.

The age at which the hair begins to turn *grey* is a variable one, the onset at one age or another being determined by hereditary predisposition. In grey horses, which are dark when quite young, the greying depends upon a dominant heredity factor.

A congenital lack of hair (*hypotrichosis*) is a hereditary disorder. G. Linzenmeier[3] has reported concerning two families in which the lack of hair was manifestly the outcome of a dominant heredity (Fig. 73). A. M. Gossage[4] has published a genealogical tree illustrating this. More common, however, are cases in which only isolated individuals, or a small number of brothers and sisters, are affected with hypotrichosis, their parents and their children having a normal growth of hair. According to C. H. Danforth,[1] we must, therefore, also suppose that there are recessive heredity factors which can give rise to lack of hair.

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[1] R. Sabourand, Corrélation entre l'évolution génitale et la pathologie du système pileux dans l'espèce humaine, "Archives mensuelles d'obstétrique et de gynécologie", vol. V, 1914, p. 1.

[2] D. Osborn, Inheritance of Baldness, "Journal of Heredity", vol. IV, no. 8, 1916.

[3] Die Vererbungsgesetze der Hypotrichosis congenita an der Hand zweier Stammbäume, Studien zur Pathologie der Entwicklung, vol. I, 1914, p. 185.

[4] The Inheritance of certain Human Abnormalities, "Quarterly Journal of Medicine", vol. I, 1908, p. 331.

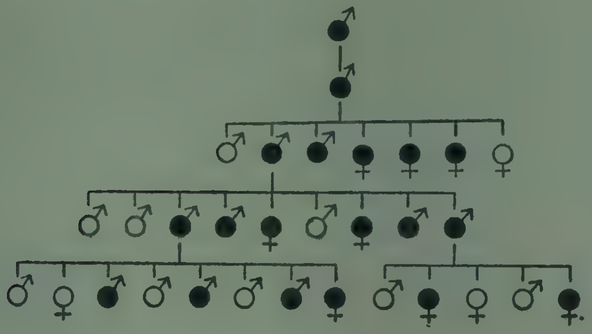

Fig. 73.
Lack of hair (hypotrichosis), after Linzenmeier. (Extract.)

E. Fischer[2] has described a family in which, in the case of several persons of both sexes, the hair of the head from youth upwards was nothing more than a thin downy growth. In the males, the beard was represented only by isolated tufts, and the rest of the body was hairless. The nails were thick and malformed; the teeth were atrophic.

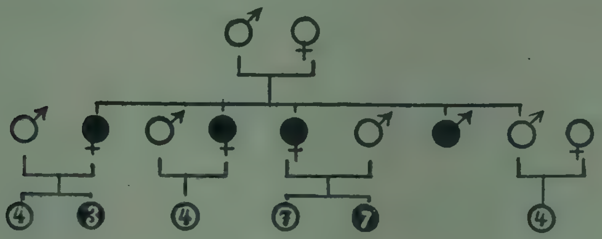

Fig. 74.
Congenital lack of hair, after E. Fischer.

This genealogical tree (Fig. 74) does not show clearly whether the anomaly was dominant or recessive. The fact that the parents were not affected, would, on the whole, make us regard it as recessive. The two marriages in the second generation from which children showing imperfect growth of hair were born, were consanguineous—and it is by no means unlikely that the marriage in the first generation was also consanguineous, since the family lived in a small Alsatian village.

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[1] The Hair, with special reference to Hypertrichosis, Chicago, 1925.

[2] Ein Fall von erblicher Haararmut, "Archiv für Rassenbiologie", vol. VII, no. 1, 1910

V. Berglund[1] has described a family in which six of the members suffered from congenital baldness as far as the scalp was concerned, though the hair in other regions was normal and the teeth and the nails were likewise normal. A bald man in his first marriage with a normal woman had five children, two of which were normal, and three bald; in his second marriage with a woman who was also normal he had five more children, three of which were normal and two bald. (Fig. 75.) It was reported that the grandparents (the father and the mother of the bald father) and the nine brothers and sisters of the bald father had had a normal growth of hair. If, as these facts seem to show, we are concerned here with a dominant heredity factor, the case must be one in which the taint appeared as a mutation in the generation of the grandparents who were themselves still free. I think, however, we cannot exclude the possibility of a recessive heredity, for we are concerned with a family in a remote rural district of Sweden, where inbreeding may very likely have led to the outcrop of a recessive anomaly.

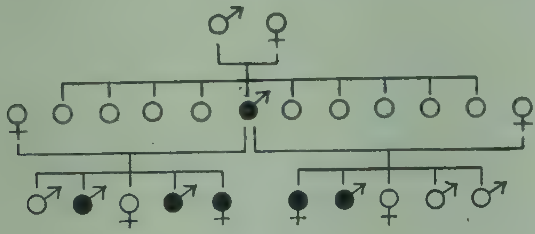

FIG. 75.
Congenital lack of hair, after Berglund.

L. Plate[2] found that in hairless dogs, the heredity of the hairlessness was dominant.

"*Hairy men*" and "*hairy women*"—persons with an excessive growth of hair on the body and the face—are very seldom encountered as members of the same family. The hairiness appears to be due to a persistence of the foetal "lanugo" or "down", and to be associated with defective development of the teeth—but little is known as to its heredity.

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[1] Sechs Fälle von Hypotrichosis in einer Familie, "Hereditas", vol. V, no. 1, 1924.

[2] Ueber Hundekreuzungen, "Verhandl. der Deutschen Zool. Ges.", 1925, p. 89.

In *moniletrichosis* ("beadiness" of the hairs) the individual hairs are thickened at intervals so that each hair resembles a string of beads, and the hair as a whole is thinned so that the individual hairs are about one millimetre apart. According to Siemens and Heuck,[1] who were able from the literature on the subject to collect information relating to sixteen families in which the affection prevailed, this anomaly has an irregular dominant heredity.

Sebaceous cysts are produced by the inversion of skin rudiments, and are generally found in the hairy scalp, where they may attain the size of a hen's egg or larger. The inverted skin rudiment continues to produce epithelial scales, sebaceous secretions, and hairs, which, finding no outlet, gradually accumulate to form the cyst. In any individual, such cysts are usually multiple, and we generally find sebaceous cysts in several members of the same family. The heredity is in most cases dominant, but the trouble need not necessarily become active in every carrier of the taint.

According to H. W. Siemens,[2] there is also a non-heredity form of sebaceous cyst, developing out of a follicular cyst.

Absence of the nails, technically known as *anonychia*, has occasionally been observed in several members of the same family. Defective development of the nails is not infrequent as a family peculiarity. Particularly common is a troublesome anomaly owing to which the fold of skin which covers the root of the nail will not separate from the nail, with the result that very disagreeable lacerations occur. This anomaly, which is apt to be associated with abnormal softness and smallness of the nails, seems to depend upon a dominant or intermediate heredity. The tendency to the formation of white patches in the nails (*leuconychia*) also appears to have a dominant heredity.

A. W. Bauer[3] has described a family nineteen members of which had marked leuconychia. Seventeen of these persons suffered also from sebaceous cysts in the hairy scalp.

[1] O. Heuck, Moniletrichosis (Siemens' Studien über Vererbung von Hautkrankheiten, VII), "Archiv für Dermatologie", vol. CXLVII, 1924, p. 196.

[2] Studien über Vererbung von Hautkrankheiten, V, Atherom, "Archiv für Dermatologie", vol. CXLIV, 1923, p. 175.

[3] Heredofamiliäre Leukonychie, "Zeitschr. für angew. Anatomie und Konstitutionslehre", vol. V, 1919, p. 47.

Extreme thickening of the nails (hyperkeratosis), sometimes conjoined with a claw-like bending of the nails (onychogryphosis), has occasionally been observed to show a dominant heredity.

Clubbing of the finger-ends, associated with a longitudinal flexion of the finger-nails, also runs in families. It is markedly correlated with pulmonary diseases, and especially with pulmonary tuberculosis. In many cases the clubbed fingers appear to be a sequel of the pulmonary disease, which is itself the expression of a family tendency;[1] but it seems that the anomaly is sometimes hereditary per se.[2]

5. ANOMALIES OF BODILY FORM

All hereditary troubles may, in a sense, be regarded as *errors of development*. In the majority of such troubles, however, the error of development is not obvious to the naked eye, but concerns the internal structure of the organs or of the minute tissue elements, the cells. Abnormal structure of the cells leads to an abnormal functioning of the affected organs. Generally when we speak of an error of development, and still more when we use the word malformation, we are not referring to a morbid structural development affecting numerous minute tissue elements, but to an obvious anomaly in external form.

Every hereditary error of development is primarily dependent upon an error of development in the hereditary equipment, and this is not itself directly perceivable. From the error of development in the hereditary equipment in the fertilised ovum, an unbroken developmental series leads to the error of development that exists in the newborn infant. Strictly speaking, therefore, we are not entitled to say that a hereditary error of development originates at any particular moment of time, and the most we can do is to say when its existence first became manifest. In common parlance, the word "malformation" is used for those deviations of form which are already obvious at the time of birth. Nevertheless, as a matter of fundamental principle, we cannot say that birth is the latest moment of time at which a malformation or an error of development can arise. The individual's development is by no means completed at the time of birth. We can, therefore, for example, speak of hereditary exostoses as malformations, although they do not exist at birth, and only develop later in life (see below, p. 297). But the general development of the bodily form and of the limbs is manifest in the embryo long before birth, three months after conception, so that most of the graver malformations have already been produced at this early period of intra-uterine life.

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[1] E. Ebstein, Angeborene familiäre Erkrankungen an den Nägeln, "Dermatologische Wochenschrift", 1919, p. 113.—Ibid. Familiäres Vorkommen von Verdickung der Endphalangen, "Medizinische Klinik", no. 52, 1920.

[2] E. Lewy, Beitrag zur Kenntnis der kongenitalen Trommelschlägelfinger, "Medizinische Klinik", no. 28, 1921.

In addition to hereditary malformations, there are non-hereditary congenital malformations resulting from disease in the mother, from the effect of poisons, or from mechanical influences. By errors in the formation of the foetal membranes, especially when the cavity they enclose is too small and when the waters are insufficient in quantity, or when cords form within the amnion, grave disturbances of development may arise, but these of course are not hereditary. Heredity could only play a part here if such malformations of the amnion were themselves hereditary—and, even then, the malformations produced thereby in the offspring would not be identical with those in the parents, for the inheritance would be more or less irregular. The formation of cords within the amnion may strangle the limbs of the foetus, with the result that a child will be born with only one arm or only one foot. Speaking generally, however, the importance of errors of amniotic development is far from being as great as used to be supposed. There is hardly a malformation which has not been referred to this cause, including a great many which are beyond question hereditary. If a malformation of the same kind occurs in numerous members of a family, we can say with certainty that it cannot be caused by an error in the development of the amnion. Furthermore, when malformations are symmetrical, there can be little doubt that they are truly hereditary. If, for instance, we find the same malformation in both hands, we can conclude with the greatest probability that the trouble is hereditary. The same thing applies to malformations which exhibit a serial homology, being of the same character both in the hands and in the feet.

The majority of unilateral malformations, on the other hand, appear to be non-hereditary, but we must not go so far as to assume that a malformation is not hereditary because it is unilateral. We even have family trees showing the hereditary transmission of an anomaly affecting one particular side. It is likely enough that in human beings, just as in certain other animal organisms, one-sided malformations may occasionally result from an abnormal distribution of the chromosomes at the time of the primary division of the fertilised ovum. In butterflies, for instance, the so-called unilateral hermaphrodites originate in this way. To Poll, moreover, we are indebted for the drawing of a bullfinch, a unilateral hermaphrodite, showing on one side of the breast the red feathers of the cock bird, and on the other side the grey feathers of the hen.[1] My own experience has shown that similar unilateral anomalies sometimes occur in butterflies as a result of the working of heredity factors that have nothing to do with the determination of sex. We may suppose, therefore, that unilateral malformations will from time to time be produced in this way in human beings.

We can define *malformations* as deviations in bodily structure, and especially in the external form of the body, which are the outcome of a developmental error and interfere with the organism's capacity for adaptation. In the present work our interest in malformations is confined to those which are hereditary. The congenital lack of fingers as an outcome of intra-uterine amputation by amniotic cords is much more akin to the accidental loss of fingers at any time after birth than it is to a lack of fingers from hereditary causes. The effects of intra-uterine accidental amputations are no more transmissible by heredity than are the effects of mutilation after birth. The heredity of the most familiar among the genuinely hereditary malformations of the limbs is, in the main, dominant.

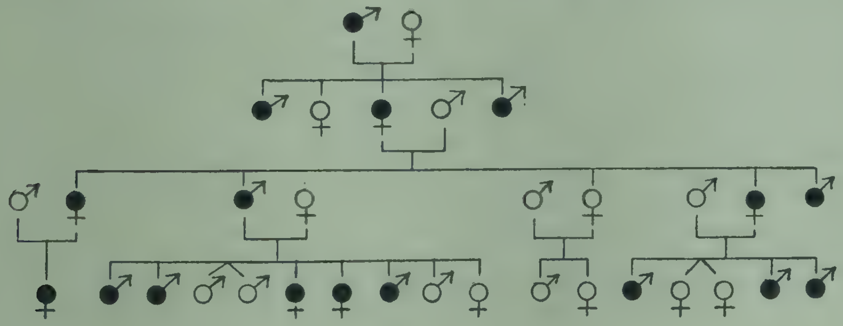

FIG. 76.
Polydactylism, after Sverdrup. (Extract.)

Let us first consider what is known as *polydactyly* or *polydactylism*, the condition in which people are born with supernumerary fingers or toes. In Fig. 76 we have a genealogical tree showing the family incidence of this malformation.

A. Sverdrup[2] describes a kinship in which 37 persons had superfluous fingers. The heredity of the anomaly could be traced uninterruptedly through six generations. It was never transmitted by normal individuals, and was therefore typically dominant (or intermediate, since we do not know what homozygotic carriers of the taint look like). The same remark applies to a family described by G. Amrain.[1] Matters, however, are not always so simple as this. In most genealogical trees illustrating the family incidence of polydactylism, we note the overleaping of generations. Yet there is no reason to suppose that the heredity is recessive, and it follows that the taint does not manifest itself in all those who carry it. O. Köhler[2] has published a family tree in which, among four polydactylous persons, only two were brother and sister, the kinship of the others being fairly remote. Again, in the genealogical tree published by Sverdrup, part of which is reproduced in Fig. 76, though it appears so regular, by no means all the polydactylous persons had twelve fingers and twelve toes. In most of them, it is true, the little finger was more or less completely doubled: but in other instances, a sixth finger existed only in the form of a little appendage; and in yet other cases one hand or one foot was perfectly normal. We infer from this that conditions during the period of development must also have some influence. Thus it was, in a sense, a mere chance that in this particular family there was no overleaping of a generation; for seeing that, notwithstanding the presence of the polydactylous taint, one hand could be normal, we might obviously expect that in some such persons both hands would likewise be normal. We see, then, that the heredity of polydactylism is irregularly dominant; that is to say, as regards the occurrence of polydactylism, a particular dominant heredity factor is not the only thing that counts, for external conditions also have an influence during the period of development—and perhaps, in addition, other heredity factors which would not per se be competent to produce polydactylism. For the rest, it is plain that in different families there may be different morbific heredity factors at work in the production of polydactylism, inasmuch as the type of the malformation varies considerably from one family to another. Comparatively often, too, we find polydactylism combined with other malformations of the hands and the feet, and in such instances we certainly have to do with a heredity factor which can cause malformations of various kinds. Examples of this are given by Köhler.

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[1] A. Moll, Handbuch der Sexualwissenschaften, third edition, Leipzig, 1926, plate viii.

[2] Postaxial Polydactylism in six Generations of a Norwegian Family, "Journal of Genetics", vol. XII, no. 3, 1922.

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[1] Ein Fall von hereditärer Hexadactylie, Dissertation, Basle, 1913.

[2] Ueber die Vererbung der Vielfingrigkeit beim Menschen, "Biologisches Zentralblatt", vol. XLIII, no. 6, 1924.

In the literature of polydactylism, we find again and again the statement that in a southern French village (Izeaux in the department of Isère) polydactylism was so widely diffused that at last the majority of the inhabitants became affected. Inbreeding was said to be the cause of this; for as soon as the inbreeding ceased, the polydactylism disappeared. R. Sommer[1] has shown that this story, for which a French writer named Potton was primarily responsible, is unfounded. Besides, inbreeding has no special effect in the case of dominant taints.

Syndactylism is a condition in which two or more fingers or toes are grown together, the most common type of this malformation being that in which the middle finger and the ring finger are thus connected. In many such families, the fingers are only connected by a web of skin; but in others the bones, too, have grown together. In a genealogical tree published by C. Schlatter,[2] syndactylism is shown to affect twenty persons in uninterrupted hereditary succession, so that the taint is, manifestly, simply dominant. In a family described by K. Vogel,[3] syndactylism in conjunction with polydactylism showed a dominant heredity.

In a family concerning which R. Schofield[4] has reported, a webbed connexion between the second and third toes was transmitted by inheritance exclusively to males, affecting every one of the male offspring. In all, fourteen males had this malformation. Such a hereditary transmission exclusively to males is theoretically conceivable, on the hypothesis that the acting heredity factor is contained in the Y-chromosome; but I regard Schofield's report as unduly condensed, and I do not think we are justified in drawing from it any far-reaching conclusions.

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[1] Ueber Familienähnlichkeit, Vienna, 1917.

[2] Die mendelschen Vererbungsgesetze beim Menschen an der Hand zweier Syndaktylie-Stammbäume, "Korrespondenzblatt für Schweizer. Aerzte", 1914, no. 8.

[3] Ueber familiäres Auftreten von Polydaktylie und Syndaktylie, "Fortschritte der Röntgenstrahlen", vol. XX, 1913, p. 443.

[4] Inheritance of Webbed Toes, "Journal of Heredity", vol. XII, no. 9, 1921.

Another malformation which has been observed to run in families is one which makes it impossible to supinate the hand, the bones of the forearm being grown together. This is known as *radio-ulnar synostosis.*[1] In many cases the manifestation of the taint is unilateral, so we may presume that in other instances it remains completely masked. The nature of the heredity has not been fully elucidated, but there are many indications that it is incompletely dominant.[2]

There are various kinds of *brachydactylism* (abnormal shortness of the fingers and toes). The form of brachydactylism (also known as brachydactylia or brachydactyly) described by W. C. Farabee[3] has become classical (Fig. 77). In this malformation the fingers have only two joints, instead of three, and the thumb only one joint instead of two. The anatomical basis of the malformation is thus *hypophalangism* or *hypophalangy*, that is to say the lack of one of the phalanges in the fingers and the thumbs. In persons thus affected, the arms and the legs and also the body as a whole are too short. Brachydactylous men are on the average 21 cm. shorter than their normal brothers, and brachydactylous women 12 cm. shorter than their normal sisters. The taint, therefore, is one which interferes with the growth in length of the bones in general. The heredity is dominant.

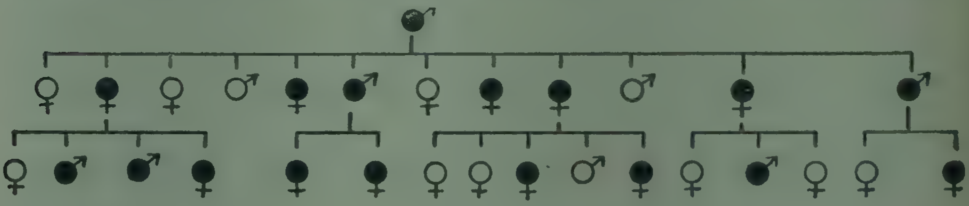

FIG. 77.
Brachydactylism, after Farabee. (Extract.)

As a matter of historical interest we should note that it was in the case of brachydactylism that the mendelian law of inheritance was first proved to apply to human beings. In one kinship, Farabee found 36 brachydactylous persons to 33 normal-fingered; in a second, the ratio was 42 : 33; in a third, it was 21 : 26. Adding these figures together we get 99 brachydactylous persons to 92 normals, which, in view of the small numbers dealt with, corresponds closely enough to the theoretical ratio of 1 : 1.

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[1] C. B. Davenport, H. L. Taylor, and L. A. Nelson, Radio-Ulnar Synostosis, "Archives of Surgery", vol. VIII, 1924, p. 705.

[2] R. Schinz, Vererbung und Knochenbau, "Schweizerische Medizinische Wochenschrift", vol. LIV, nos. 50 and 51, 1924.

[3] Inheritance of Digital Malformations in Man, "Papers of the Peabody Museum", vol. III, no. 3, Harvard University, 1905.

A slighter form of brachydactylism has been described by H. Drinkwater[1] under the name of "*minor-brachydactyly*". Here the fingers have the normal number of joints, but the joints are very short. Pfitzner points out that this malformation would be more aptly described as *brachyphalangism*. Its heredity, likewise, is dominant. Just as little as in the case of hypophalangism, do we know in the case of brachyphalangism what people carrying the taint homozygotically would look like.

Another kind of brachydactylism has been described by the Norwegian investigators O. L. Mohr and C. Wriedt.[2] They were able to trace the recurrence of abnormal shortness of the middle joint of the index finger for six generations. The anomaly presented itself in two obviously different degrees, so that the observers were led to believe that, in addition to the primary heredity factor, a secondary heredity factor was sometimes at work. Two female mon-oval twins exhibited precisely the same degree of brachyphalangism. When a generation was apparently overleaped, X-ray examination showed, nonetheless, a plain shortening of the bone. The authors rightly point out that in many cases it is only because of inadequate examination that a generation is supposed to have been overleaped. Hanhart has observed what appears to have been the same kind of hereditary brachyphalangism in the members of a Swiss family. Mohr and Wriedt, in their study of the subject, distinguish between nine different kinds of brachydactylism, all of which appear to have a dominant heredity.

When two of the joints of the same finger have grown together, we speak of *symphalangism* or *orthodactylism*. The commonest lack of an inter-phalangeal articulation is that of the second articulation of the middle finger. When this malformation is present, the hand cannot be properly closed. The extent to which symphalangism occurs may vary to a considerable degree within any one affected family, sometimes only a single articulation being absent, and sometimes several articulations. Here, likewise, a generation may occasionally be skipped. In the genealogical trees published by Walker[1] and K. Morgenstern,[2] the heredity was regularly dominant.

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[1] Account of a Family showing Minor-Brachydactyly, "Journal of Genetics", vol. II, 1912, p. 21.

[2] A New Type of Hereditary Brachyphalangy in Man, Carnegie Institution of Washington, 1919.

In what is known as *clinodactylism*, the terminal joint of the little finger and often the terminal joint of the ring finger as well are inclined towards the middle finger, and are stiff. The heredity appears to be dominant.[3] A lateral deviation of fingers may also arise as an outcome of brachyphalangism of particular fingers.

In what is termed *camptodactylism*, the little finger and usually the ring finger as well are fixed in a position of flexion. In a family studied by S. Goldflam,[4] twenty-six persons in three generations were thus affected.

The malady known as *Dupuytren's contracture*, which consists of a shortening and stiffening of the tendons and ligaments of the little-finger side of the palmar surface of the hand, only begins in adult life. It occurs chiefly in males, and in a number of families its occurrence can be traced for several generations. The taint appears to be simply dominant. Occupational injuries make it worse.

Hammer-toe, in which the last joint of a toe becomes fixed in flexion, has been found by J. Paulsen[5] and M. J. Gutmann[6] to run in families. In the family tree published by Gutmann, in the seven persons who were affected the deformity developed only on the second toe of the right foot, thus giving an example of the inheritance of a unilateral anomaly persisting always on the same side of the body.

What is known as *split-foot* is a serious hereditary malformation in which the anterior part of the foot is divided into two sections, each of them bearing one or several toes. T. Lewis has published six genealogical trees, all showing a dominant heredity.[7] In persons suffering from split-foot, the hands likewise are as a rule more or less malformed, there being sometimes split-hand, and sometimes a lack of one or more fingers (*ectrodactylism*), which may go so far that only one finger remains. In members of the same affected family, the extent of malformation of the hands and the feet may vary to a considerable degree. In a family described by L. R. Grote,[1] there were peculiar and irregular intermediate forms of split-foot, syndactylism, and polydactylism. The heredity factor at work in these cases had produced irregular disturbances of the mechanism of division which leads to the formation of the fingers and the toes.

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[1] Quoted by R. Schinz, Vererbung und Knochenbau, "Schweizerische Medizinische Wochenschrift", vol. LV, nos. 50 and 51, 1924.

[2] Ueber kongenitale hereditäre Ankylosen der Interphalangealgelenke, "Beiträge zur Klinischen Chirurgie", vol. LXXXII, 1913, p. 508.

[3] K. Wegelin, Ueber eine erbliche Missbildung des kleinen Fingers, "Berliner Klinische Wochenschrift", 1917, p. 283.

[4] Ein Fall von kongenitaler familiärer Ankylose der Fingergelenke, "Münch. Med. Wochenschr.", no. 47, 1906.

[5] Kasuistische Beiträge zur Vererbungslehre, "Archiv für Rassenbiologie", vol. XVI, no. 3, 1925.

[6] Zur Vererbung der Hammerzehe, "Archiv für Rassenbiologie", vol. XVII, no. 2, 1925.

[7] Split-foot, "Treasury of Human Inheritance", parts 1 and 2, Cambridge University Press, 1909.

Hereditary split-hand is extremely rare. A genealogical tree published by R. Fetscher,[2] suggests a recessive heredity.

The name of *club-foot* (*pes varus*) is given to a malformation owing to which the feet are bent strongly inward, so that the soles look towards one another, and in walking, instead of the sole of the foot, what should be the outer margin of the foot touches the ground. At the same time, the point of the foot is usually bent downwards. In every thousand births, there is approximately one infant with club-foot, males being twice as common as females. In about half the cases, both feet are affected.

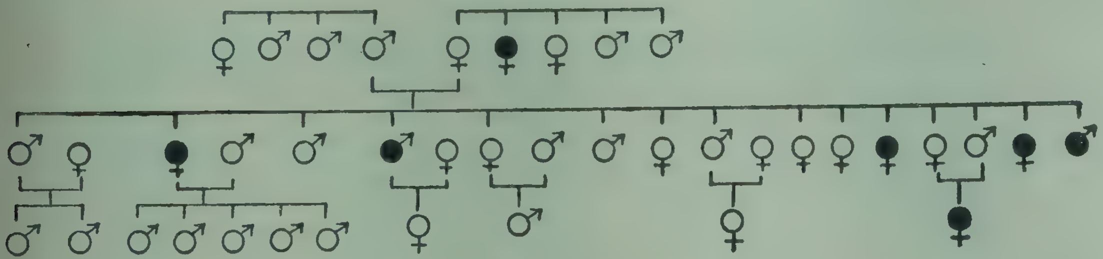

FIG. 78.

Club-foot, after Fetscher.

The club-footed girl in the third generation is the offspring of incest between brother and sister.

R. Fetscher,[3] having examined the material at the Tübingen Surgical Clinic, comes to the conclusion that the heredity of club-foot is recessive. It would appear, however, that external influences play some part in causing the malformation. As a possible exciting cause, we have to think of an unfavourable position of the foetus in utero, such as may arise when the foetal membranes are too small. Club-foot is not infrequently associated with malformation of the spinal cord, and it therefore seems possible that a malformation of the nervous system may occasionally give rise to club-foot.

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[1] Ueber vererbliche Polydaktylie, "Zeitschrift für Konstitutionslehre", vol. IX, 1924.

[2] Ein Stammbaum mit Spalthand, "Archiv für Rassenbiologie", vol. XIV, no. 2, 1922.

[3] Ueber Erblichkeit des angeborenen Klumpfusses, "Archiv für Rassenbiologie", vol. XIV, no. 1, 1922.

As already said, club-foot is twice as common in males as in females. We do not know the explanation of this difference between the sexes. Some of the family trees published by Fetscher are compatible with the theory that the heredity is recessively sex-linked—as we see, for instance, in Fig. 79. It is, however, also possible that a heredity factor transmitted equally to the two sexes is (for some unknown reason) especially inclined to become manifest in males.

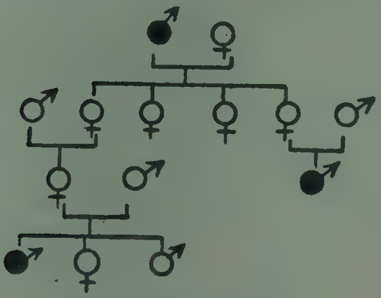

FIG. 79.
Club-foot, after Fetscher.

A genealogical tree published by J. Kochs[1] suggests, rather, that the heredity of club-foot must be dominant. Here in three generations there were eleven individuals affected with club-foot. Some genealogical trees compiled by O. Müller out of the material of the Zurich Surgical Clinic likewise favour the notion of a dominant heredity. It is possible that in different families club-foot may be caused by different heredity factors.

The condition known as *flat-foot* (*pes planus*), in which the arch of the foot gives way and the foot bends outwards (*splay-foot*), would seem in most cases to arise as the outcome of a hereditary taint. Genealogical trees compiled by Dr. Hubert Waldmann and personally communicated by him to the present writer favour the notion that the heredity is dominant. Flat-foot appears to be especially common in Jews. During the Great War, R. N. Salaman, examining 5000 Jewish soldiers, found flat-foot in about one sixth, whereas this defect existed in about one fortieth only of other soldiers in the British army.[2]

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[1] Ueber die Vererblichkeit des angeborenen Klumpfusses, "Münchener Medizinische Wochenschrift", vol. LXXI, no. 35, 1924.

[2] Eugenics in Race and State, vol. II of the Transactions of the Second International Congress of Eugenics, Baltimore, 1923.

It would seem that hereditary predisposition must play a considerable part in producing a deviation of the great toe towards the mid-line of the foot (*hallux valgus*), which in former days was attributed exclusively to the pressure of ill-fitting shoes. Hanhart informs me that he has been able to compile a family tree showing sixteen persons affected with this anomaly in four generations, and making it plain that the heredity was dominant. Several members of this family who had never worn pointed shoes showed a very marked degree of this deviation.

Hereditary predisposition also plays a considerable part in producing *knock-knee* and *bow legs* (sometimes termed *X-legs* and *O-legs* respectively). H. Ziesch[1] has published some genealogical trees which are very instructive in this respect. In former days it was supposed that such malformations of the knee joints were mainly due to prolonged standing, though sometimes rickets was invoked as well to account for the trouble. Recently, however, it has been found that rickets (of which malady, too, heredity is a part cause, as will be shown below on p. 333) produces X-legs or O-legs, as the case may be, only in certain families. W. Weitz,[2] in his study of twins, found eight pairs of mon-oval twins with X-legs and four pairs with O-legs developed to an equal degree in the two members of each pair. Still, there can be no doubt that prolonged standing is in many cases a contributary cause of the development of X-legs, so that in Germany knock-kneed persons are often said to have "bakers' legs".

Hyperextensibility of the joints, due to a general slackness of the ligamental apparatus or parts thereof, and especially hyperextensibility of the fingers, would appear in some instances, according to the observations of E. Ebstein[3] and Hanhart, to manifest in some cases a dominant heredity. Hanhart informs me that he has been able to compile a family tree showing this peculiarity in twenty members of the kinship.

Congenital dislocation of the radius with shortening of the ulna

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[1] Statistisch-genealogische Untersuchungen über die Ursachen der Rächitis, "Archiv für Rassenbiologie", vol. XVII, no. 1, 1925.

[2] Studien an eineiigen Zwilligen, "Zeitschrift für Klinische Medizin", vol. CI, nos. 1 and 2.

[3] Klinische Beobachtungen über Vererbung von Krankheiten, "Archiv für Rassenbiologie", vol. XV, no. 1, 1923.

(radio-ulnar olisthia) had a dominant heredity in a family tree published by L. C. Thomas.[1]

Congenital dislocation of the hip, a condition in which the head of the femur is no longer in the acetabulum, is certainly in part due to hereditary predisposition. The malformation, which is not very rare, is six times as common in females as in males. Often the trouble develops only on one side, and this leads us to suspect that the taint may not manifest itself in all persons in whom it is present, but that environmental influences (operative before birth—the position of the foetus in utero) may act as exciting causes.

G. Roch[2] compiling a number of genealogical trees out of the material of the Tübingen Surgical Clinic, infers that besides recessive heredity factors there may in exceptional cases be dominant heredity factors giving rise to congenital dislocation of the hip. The genealogical tree reproduced in Fig. 80 seems to me, rather, to suggest an irregularly dominant heredity. The fact that a man suffering from congenital dislocation of the hip marries two women free from the malformation and with each of them procreates offspring suffering from it, is hardly compatible with the idea of a recessive heredity. Consanguineous marriage does not appear to be more frequent than the average in the parents of persons suffering from congenital dislocation of the hip, as we should expect if the heredity were recessive. The Norwegian anthropologist H. Bryn has drawn attention to evidence showing that congenital dislocation of the hip is especially common in areas where there is an unusually intense mingling of races (Lapland, Japan, Bohemia). If this be so, the malformation may be dependent upon an incompatibility of the

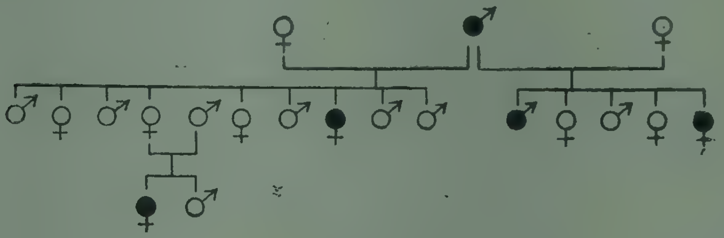

Fig. 80.
Congenital dislocation of the hip, after Roch.

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[1] Five Studies in Human Heredity, "Eugenical News", vol. XI, no. 10, 1926.

[2] Die Vererbung der sogenannten angeborenen Hüftverrenkung, "Archiv für Rassenbiologie", vol. XVII, no. 3, 1925.

genes of different races—in a word upon polymery. Hooff has published a study of the subject in the "Archiv für Rassenbiologie", 1927. In twin sisters who were manifestly mon-oval, only one of the pair had this complaint, a circumstance indicating the cooperation of environmental influences.

Perthes' disease (*osteochondritis deformans coxae juvenilis*) an affection of the hip-joint resulting from defective formation of the bones and cartilages of the part, is sometimes observed in several members of the same family.[1] There is likewise a family proclivity to *arthritis deformans*, a disease which comes on in middle life or in old age, in which the joints become thickened and painful, the articular surfaces being roughened and fibrillated. Weitz's studies of twins lead him to the conclusion that this form of arthritis is the outcome of a hereditary predisposition; and that when the disease appears to result from hard work and exposure to cold, it is only because these act as exciting causes.

Absence of the patella, apparently with a dominant heredity, has been observed in certain families.[2]

Among the hereditary malformations of the limbs may be mentioned *multiple enchondromata* and *exostoses* (cartilaginous and bony outgrowths), although these are not present at birth. They usually make their appearance during the years of growth, varying much in size, and being apt to deform the hands and to interfere with the functions of these. When the growth of the skeleton ceases, such outgrowths likewise cease to enlarge, this showing that they are true malformations. A collection of 184 genealogical trees illustrating the family prevalence of the affection shows that its heredity is dominant.[3]

The defect known as *hypospadias* is a markedly hereditary

[1] W. Müller, Beobachtung zur Frage des Verlaufes, der Endansgänge sowie des familiären Auftretens der Osteochondritis deformans coxae juvenilis, "Archiv für orthopädische Unfallchirurgie", vol. XX, 1922, p. 327.

[2] Wuth, Ueber angeborenen Mangel der Kniescheibe, "Archiv für klinische Chirurgie", vol. LVIII, 1899.—Wolf, Zwei Fälle von angeborenen Missbildungen, "Münch. Med. Wochenschrift", 1900, no. 22.—W. Scheidt, Erbliches Fehlen der Kniescheibe, "Anthropologisch. Anzeiger", vol. II, no. 1, 1925.

[3] P. Stocks and A. Barrington. Hereditary Disorders of Bone Development, "Treasury of Human Inheritance", vol. III, part I, London, 1925.

malformation of the generative organs. This is an affection peculiar to males, in which the aperture of the urethra, instead of being at the end of the penis, is on the posterior surface of that organ at a varying distance from the extremity. About one in three hundred of all males exhibits this malformation to a greater or less extent. In certain families its inheritance can be traced in the male line through several generations. From the nature of the case, it cannot affect females, but it can be transmitted by healthy women to their male children. This type of inheritance must not be confounded with the recessive sex-linked heredity discussed above on p. 225. The heredity of a dominant taint which cannot possibly manifest itself in females differs from the heredity of a recessive sex-linked taint in this respect, that in the latter the malformation cannot be directly transmitted from father to son, whereas in the former it can be.

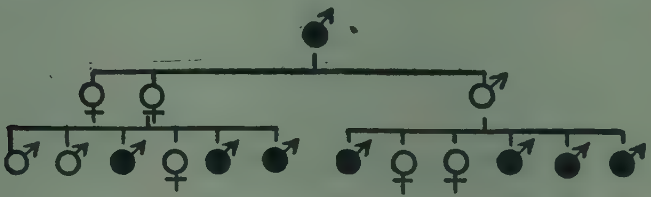

FIG. 81.
Hypospadias, after Lesser. (Extract.)[1]

W. Bulloch records a number of genealogical trees, not all of which, however, are founded upon trustworthy data.[2] Manifestly in the case of such a trouble as this, about which people are apt to be secretive, it is exceptionally difficult to compile trustworthy family trees. Those which we have, produce the impression that there must be several heredity factors giving rise to hypospadias, this explaining why it varies in severity in different families.

Most instances of what is called *pseudohermaphrodism* are nothing more than cases of high-grade hypospadias. The scrotum is split in two; the penis is very small, or may be entirely absent. Persons with this malformation are often wrongly supposed to be girls until the error becomes manifest at puberty. Numerous cases of the kind are recorded in the Treasury of Human Inheritance. Inasmuch as such high-grade hypospadias is generally to be observed only in one generation of brothers, we are naturally inclined to assume that its heredity is recessive. This would be in conformity with the general principle that high-grade anomalies are apt to be recessive, and low-grade anomalies dominant.

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[1] E. Lesser, Beitrag zur Vererbung der Hypospadie, "Virchows Archiv", vol. LXIV, 1889, p. 537.

[2] Hereditary Malformation of the Genital Organs, "Treasury of Human Inheritance", part III, London, 1909.

Since in man, likewise, the development of the sex characters is determined by the hereditary equipment, we are justified in assuming that the (very rare) cases of *true hermaphrodism* in human beings develop upon a hereditary foundation. We speak of true hermaphrodism when both male and female reproductive glands are present. When an individual has exclusively male or exclusively female reproductive glands in association with the external characters proper to the opposite sex, we speak of *pseudo-hermaphroditismus masculinus vel femininus*. Those kinds of pseudo-hermaphrodism in which masculine and feminine sexual characters are variously mingled, are beyond question hereditarily determined. In certain butterflies, Goldschmidt has been able, by combining the heredity factors of different races, to produce intersexual individuals of various kinds (gynanders, etc.). It is not improbable that similar intersexual types can be produced in human beings by a combination of abnormal genes. According to O. Polano,[1] who has described a recent case of true hermaphrodism, hitherto only four cases of true hermaphrodism have been studied in the human species.

The testicles, or male reproductive glands, are formed within the abdominal cavity of the embryo to descend (normally) into the scrotum during intra-uterine life. When they remain in the abdominal cavity, or when their descent is arrested in the inguinal canal, we speak of the person thus affected as a *cryptorchid*. Heredity certainly plays a considerable part in the causation of *cryptorchism*. As a rule, in such cases, only one of the two testicles remains in the abdominal cavity or in the inguinal canal. The malformation is not to be regarded as a perfectly harmless one, for a testicle arrested in the inguinal canal cannot develop normally, being liable to be injured by pressure, and subject to inflammatory changes. In a notable proportion of all male infants, the descent of the testicles is not completed by the time of birth, but in such cases descent is usually completed during childhood. It is probable that undue smallness or complete absence of the testicles is hereditarily determined, but we have no trustworthy information about the matter.

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[1] Ueber wahre Zwitterbildung beim Menschen, "Zeitschrift für Geburtshilfe und Gynäkologie", vol. LXXXIII, no. 1.

In connexion with disturbances of the descent of the testicles, the hereditary liability to *rupture* may suitably be considered, rupture in males depending to a large extent upon imperfect closure of the inguinal canal after the descent of the testicle. The actual onset of rupture is in most cases a gradual one, resulting from external causes, especially from the effort of lifting heavy weights, when part of the peritoneum and usually one or more coils of intestine as well are forced down through the inguinal canal. About 1 in every 20 or 30 men is ruptured, whereas only about 1 in 150 women is ruptured. In any case, a rupture involves a considerable impairment of the working powers, and may imperil life by becoming strangulated or inflamed. For these reasons, the question as to the hereditary character of the predisposition to rupture is of practical moment. Rupture is unmistakably one of the complaints which tend to run in families, and we derive the impression that we have to do with dominant heredity factors which (as necessarily in the case of hypospadias) do not usually manifest themselves in females. A general study of the data leads us to suppose that in the origination of rupture, heredity must play at least as important a part as do the exciting causes of the trouble. In mon-oval twins, *inguinal hernia* has several times been observed in both members of a pair—by Weitz, for instance.

A comparatively harmless hereditary malformation is an abnormal constriction of the male foreskin, the condition known as *phimosis*, which is often associated with undue length of the foreskin.

Turning to consider malformations peculiar to the female reproductive organs, we find that in large measure they are hereditarily determined. This applies to the not infrequent anomalies in the shape of the uterus (different degrees of reduplication, etc.); and also to defective development of the reproductive organs in general (see Infantilism, p. 311). The occurrence of *supernumerary breasts* or *supernumerary nipples* can be counted among hereditary malformations. The occurrence of supernumerary nipples can be traced through several generations of a family. Excessive development of the mammae in men can be classed with pseudohermaphrodism.

Hereditary causation must also be at work to produce what is known as *epispadias*, a rare condition in which the anterior surface of the penis is split, and in which the fissure may extend to include the anterior belly-wall and the bladder. Since children born with this malformation perish, there can be no question of a dominant heredity. According to H. W. Siemens,[1] epispadias is occasionally met with in both members of a pair of twins.

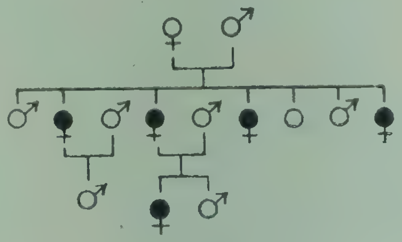

FIG. 82.
Harelip (Eugenics Record Office).

In the mid-line of the face we not infrequently see the result of a hereditary failure to close the fissure that exists here in early embryonic life. *Harelip*, for instance, consists of a deep grooving or complete splitting of the upper lip in the middle line. In severe cases, the fissure may extend between the two bones which combine to form the upper jaw; and, in the worst cases of all, the whole palate may be split likewise. On the other hand, the soft palate or the soft parts of the roof of the mouth can be cleft without any cleft in the bony palate or the upper lip. I have to thank Dr. Franz Baumeyer of Dresden for a report concerning the hereditary transmission of *cleft palate* confined to the soft structures. Cleft palate and harelip, like so many other defects of development, are, as previously indicated, the outcome of a premature arrest in the developmental process. It would seem that, according as other conditions vary, one and the same heredity factor may, during embryonic development, give rise to this arrest of development in varying degrees in different individuals; and in some persons the factor, though present, may have no manifest effect whatever. In this way, one or more generations may be skipped. As we learn from the study of the matter in the "Treasury of Human Inheritance", very few comprehensive genealogical trees illustrating the heredity of this malformation have as yet been compiled.[1] I reproduce here (Fig. 82 and Fig. 83) two family trees compiled by the Eugenics Record Office in Cold Spring Harbor in the New York State under the management of Professor C. B. Davenport.[2] In the family shown in Fig. 82, the cases were of harelip only, numbering five in all. In the family shown in Fig. 83, there were seven cases of cleft palate, and one case of harelip. According to Siemens,[3] pairs of twins have occasionally been observed showing a cleft of the hard palate of exactly the same degree and in exactly the same situation; and other pairs in which the malformation in one member of the pair was like a mirrored reproduction of the malformation in the other.

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[1] Die Zwillingspathologie, Springer, Berlin, 1924.

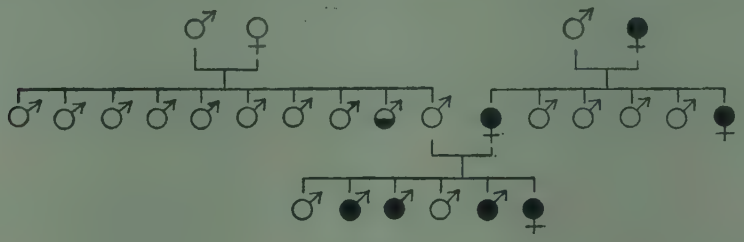

FIG. 83.
Cleft palate. (Eugenics Record Office.)
The man in the second generation indicated by a half-black circle had only a harelip without a cleft palate.

A gap between the upper central incisors (*trema*, or *diastema*) is apt to occur in several members of the same family. When present in both members of a pair of mon-oval twins, the development of the trouble is usually but not always the same in both.

McQuillen[4] has observed the absence or imperfect development of the upper lateral incisor teeth in three generations of one family. The anomaly would seem not to be exceedingly rare. T. Fürst[1] has reported on a family in which in one of the grandfathers all the incisor teeth were lacking, and there was the same defect in one grandson, while in three other grandsons the lateral incisor teeth only were lacking. L. C. Thomas has described a family in seven members of which the lateral incisors were lacking.[2]

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[1] H. Rieschbieth, Harelip and Cleft Palate, "Treasury of Human Inheritance", part IV, 1910.

[2] These genealogical trees were communicated to the Second International Congress for Racial Hygiene at New York in the year 1921; and they were reprinted in Eugenics, Genetics, and the Family, Baltimore, 1923.

[3] Die Zwillingspathologie, Springer, Berlin, 1924.

[4] Quoted by W. Praeger, Die Vererbungspathologie des menschlichen Gebisses, "Zahnärztliche Rundschau", 33rd year of issue, nos. 44 and 45, 1924.

Undue prominence of the lower jaw (lower-jaw prognathism, or *progenia*) apparently has a dominant heredity, as we see, for instance, in the Habsburg family.[3] According to A. Kantorowicz,[4] progenia occurs in about 1·5 % of the population. Kantorowicz also tells us that abnormal smallness of the lower jaw tends to run in families. In mon-oval twins, the "bite", that is to say the way in which the two rows of teeth oppose one another in the act of biting, is, according to Praeger,[5] almost always the same. The study of twins has also shown that anomalies in the position of the teeth are for the most part hereditarily determined. Moreover, according to W. Weitz's[6] observations on mon-oval twins, in any pair of these even *caries* (the process of decay which in most people if they live long enough destroys a few or many of the teeth) tends to affect exactly the same teeth. This leads us to infer that heredity must play a considerable part in originating dental caries. We must suppose that, owing to defects in the genes, the teeth have weak spots in their enamel, where the resistance to the exciting causes of caries is inadequate. During the second decade of life, a few years after the eruption of the permanent teeth, caries usually begins at these weak spots. When the areas first affected with caries are skilfully treated, the teeth in general remain immune or comparatively immune to further attacks of the disorder. The position of the teeth likewise plays a part in the origination of caries. The crowding of the teeth which certainly favours the onset of caries would seem to depend mainly upon the conjuncture of heredity factors that produce large teeth with heredity factors that produce small jaws. Racial crossing appears to play a part here. Smallness of the jaws is also associated with the adenoid constitution (see below, p. 329)—and this matter is likewise of importance in the causation of dental troubles.

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[1] Der Erbgang bei Anodontie, "Archiv für Rassenbiologie", vol. XVI, no. 3, 1925.

[2] Five Studies in Human Heredity, "Eugenical News", vol. XI, no. 10, 1926.

[3] A. Kantorowicz, Die Progenie und ihre Vererbung, "Deutsche Monatschrift für Zahnheilkunde", 1915, no. 3.

[4] Ibid.

[5] Op. cit.

[6] Ueber die Bedeutung der Erbmasse für das Gebiss nach Untersuchungen an eineiigen Zwillingen, "Deutsche Monatsschrift für Zahnheilkunde", 1924, no. 5.

It seems probable that heredity also plays a part in the causation of *pyorrhoea*, which so often leads to loss of the teeth.

Abnormal smallness of the head (*microcephaly*) has frequently been observed in several members of the same family.[1] Extreme microcephaly, which is always associated with high-grade feeble-mindedness, can apparently be caused by recessive heredity factors. C. Bernstein[2] has described a family in which, out of ten brothers and sisters, five were extremely microcephalic and feeble-minded. J. C. Frets[3], in his study of the heredity of the shape of the head, found also that microbrachycephaly of a non-morbid degree appeared to depend upon recessive heredity factors.

Tower skull (*pyrgocephaly, oxicephaly*) has been observed in several members of the same family.[4] Some of the genealogical trees illustrating the family incidence of this condition suggest a dominant heredity, whereas others suggest a recessive heredity. It is obvious that we are here concerned with a number of different anomalies. I am indebted to Dr. C. J. Caesar for a report upon hereditary tower skull. The photograph of a male child shows an extreme projection of the skull forwards and upwards, the front of the forehead being markedly in advance of the plane of the eyes. The boy has become blind owing to pressure upon the optic nerves, and his mother is blind for the same reason. Besides his mother, one of his sisters and his maternal grandmother had tower skulls in an extreme form. H. W. Siemens[5] reports that in some cases he has observed tower skulls in one member of a pair of mon-oval twins. But, from the illustrations he gives, I should say, rather, that his cases are those of a non-morbid but exceptional height of the head (*hypsicephaly*), from which it would be better to withhold the name of tower skull. Gänsslen had under observation a peculiar form of hypsicephaly associated with the haemolytic diathesis (see below, p. 367).

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[1] Goldbladt, Bruchstücke zur Kenntnis der familiären Mikrozephalie, "Archiv für Psychiatrie", vol. LXX, no. 4, 1924.

[2] Microcephalic people, sometimes called "Pinheads", "Journal of Heredity", vol. XIII, no. 1, 1922.

[3] Heredity of the Head Form in Man, "Genetica", vol. III, 1921.

[4] A. Peiper, Ueber den Turmschädel, "Monatsschrift für Kinderheilkunde", vol. XXV, 1924, p. 509.

[5] Zur Aetiologie des Turmschädels, "Virchows Archiv", vol. CCLIII, no. 3, 1924.

Complete absence of the head (*acrania*) or of the brain (*anencephaly*) occurs as a congenital malformation. In this connexion I may also mention *cyclopia*, in which the two eye rudiments have coalesced to form a functionless structure in the mid-line of the face. Obviously the heredity of this malformation cannot be dominant, but must be recessive. Inasmuch as the infants affected with it are not viable, observers hitherto, influenced by too narrow a conception of heredity, have failed to take into consideration the hereditary determination of such malformations. When people talk of "hereditary transmission," they are too apt to think only of the transmission of the qualities of the progenitors to the offspring. That is why little attention was at first paid to the tendency of such malformations in non-viable infants to run in families.[1] Had the matter been more carefully studied, we should presumably know more about it.

In the region of the vertebral column and the spinal cord there may be arrests of development in consequence of which fissures remain, through which hernia-like projections are often thrust from the lower part of the spine (*spina bifida*). Sometimes spina bifida has been observed in several members of the same family. The anomaly is rather apt to be associated with club-foot.

What is technically known as *dysostosis cleidocranialis* is a hereditary malformation of the skeletal system in which the collarbones are lacking and the head is abnormally formed. The heredity factor appears to be dominant. Schinz (op. cit.) noted this anomaly in a grandmother, a mother, and two children. In the "Treasury of Human Inheritance", several genealogical trees illustrating the malformation have been published by Stocks and Barrington.

A *dysostosis craniofacialis* having an apparently dominant (or intermediate) heredity has also been described.[2]

Water-on-the-brain (technically known as *hydrocephalus*) depends upon the presence of abnormally large quantities of fluid in the interior spaces (ventricles) of the brain, whereby the head as a whole is made excessively large. Since the anomaly has several times been observed in both members of a pair of twins, and occasionally in brothers and sisters apart from twins, there would seem to be a hereditarily determined hydrocephalus.[1] More often, however, hydrocephalus is the result of inflammatory processes (syphilis, for instance), or of poisons (such as lead).

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[1] Klopstock, Familiäres Vorkommen von Zyklopie und Arrhinanzephalie, "Monatsschrift für Geburtshilfe und Gynäkologie", vol. LVI, 1921.

[2] R. Bing, Gehirn und Auge, second edition, Munich, 1923.

A harmless hereditary anomaly is *adhesion of the lobule of the ear* to the side of the head; in more advanced cases of this defect the lobule of the ear is completely wanting. Adherent lobules have been regarded as stigmata of degeneration, and have been said to occur with especial frequency in criminals and the insane. But the same malformation is certainly found often enough in normal individuals and in perfectly healthy families. There are many parts of Germany where it occurs in one fourth of the population. R. Carrière[2] thinks we are concerned here with a dominant character. But when a malformation is so frequent as this, we should naturally be able often enough to trace it through a number of generations, even if it were recessive; and, in actual fact, the exhaustive investigations of K. Hilden[3] have shown with a high degree of probability that adhesion of the lobule of the ear or the lack of the lobule depends upon a recessive heredity factor. Hilden, however, lays stress on the fact that there are innumerable gradations between adhesion of the lobule and its absence. Furthermore, since we are concerned not with morbid anomalies but with normal variations, polymeric causation is probable. In the two members of a pair of mon-oval twins, the formation of the external ears is always very closely alike.

In connexion with anomalies of bodily form, the *habit of body* or *constitutional type* is of special importance. Anomalies connected with this are often spoken of as "constitutional anomalies". The notion of a constitutional anomaly is, indeed, as hard to delimit sharply as is that of malformation; but, nevertheless, the one, like the other, is very serviceable in practical use. Thus we speak of a "weak" or of a "strong" constitution, signifying by these terms an individual's general power of resisting noxious influences, diseases, and the effects of great exertion. In many cases a clue to the constitution is given by the external appearance, by the habit of body. Those constitutional anomalies which find expression, not so much in the habit of body as in certain functional peculiarities, those that is to say which are mainly rooted in the chemical and physiological peculiarities of the tissues, are usually spoken of as *diatheses*. These will be considered in the next subsection.

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[1] Siemens, Zwillingspathologie.

[2] Ueber erbliche Ohrformen, insbesondere das angewachsene Ohrläppchen, "Zeitschrift für induktive Abstammungslehre", 1921.

[3] Ueber die Form des Ohrläppchens beim Menschen und ihre Abhängigkeit von Erbanlagen, "Hereditas", vol. III, no. 3, 1922.

Constitutional anomalies shade into malformations, disorders of metabolism (the processes of tissue change), disturbances in the functioning of the endocrine glands, and the susceptibilities to particular diseases. The notion of "constitution" is, like the notion of "disease", directed towards the chances of self-preservation. But whereas the idea of disease already implies that the probability of self-preservation is considerably reduced, the idea of constitution is neutral in this respect, and we must qualify the term by some such adjective as "weak" or "strong" before it has any definite significance in this connexion. We speak also of a "healthy" constitution. These qualifying adjectives relate mainly to the powers of resistance that characterise the particular constitution in a very general sense. When we are referring to the special liability to some particular disease, we do not speak of a "weakness of the constitution", but of a "disposition" or a "predisposition".

I consider that J. Bauer makes a mistake when he uses the word constitution as "a general expression for all the trends incorporated in an individual's hereditary equipment".[1] This definition does not correspond in any way with ordinary linguistic usage, from which there seems to be no occasion to depart here. Nor does Bauer himself find it possible to stick to his own definition, since he characterises the particular constitutional anomalies, not in accordance with the heredity factors that underlie them, but morphologically and functionally. Still more unsatisfactory is the definition of J. Tandler,[2] who proposes to denote by the term constitution the sum of all the qualities established in the individual at the time of fertilisation "apart from the qualities of the species and the race". Such a distinction seems to be unattainable alike in theory and in practice; and Tandler's whole classification of the habit of body into "constitution" and "condition" is moreover incompatible with his own Lamarckian fundamental outlook—since Lamarckism does not, properly speaking, permit of any such severance. It is unfortunate that Tandler has secured so large a following.

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[1] Vorlesungen über allgemeinen Konstitutions- und Vererbungslehre, second edition, Springer, Berlin, 1923.

[2] Konstitution und Rassenhygiene, "Zeitschrift für angewandte Anatomie und Konstitutionslehre", 1913.

The classical instance of a constitutional anomaly is what is known as *asthenia* or the *asthenic constitution.* Persons of this type are slenderly built, with a long body and a narrow and usually flat thorax. The heart is for the most part small and weak. As an outcome of the general slenderness and narrowness of bodily structure, the viscera (heart, stomach, etc.) incline to be pendent rather than transversely disposed. The voluntary muscles, too, are weak and slack. The spine is not stiff and straight, for the asthenic "holds himself badly", not so much from neglect as because the intervertebral joints and the muscles of the back are flabby. The asthenic is very easily fatigued, alike by bodily and by mental exertion. The slackness of the digestive organs impairs nutrition. With this, too, is connected a poverty of blood, taking the form rather of a deficiency of quantity than of a thinness of the vital fluid. The reproductive glands, too, are often inadequately developed.

Owing to this primary weakness of constitution, the asthenic has less power of resisting diseases than has the normally built human being; and inasmuch as tuberculosis plays so large a part among the diseases affecting our population, an unduly large proportion of asthenics are victims of consumption.

The asthenic constitution, owing to its great frequency and to its extreme importance as leading to impairment of health and functional capacity, is one of the most notable of all bodily anomalies. "General weakness of body" which is one of the most frequent causes of unfitness for military service, and which is mainly decided by the ratio between chest measurement and stature, is usually an expression of asthenia.

On the other hand, persons with an asthenic constitution have a below-average liability to certain diseases such as diabetes, gout, obesity, pulmonary emphysema, and hardening of the arteries with its varying consequences among which paralytic strokes take the first place. Persons of the pyknic (stout and stumpy) type contrast so strongly with asthenics in the last-named respect that the pyknic type has sometimes been spoken of as the apoplectic habit of body.

Heredity is beyond question the main cause of asthenia. The asthenic habit of body is already recognisable in infancy;[1] but it does not as a rule become plainly manifest until towards the beginning of the second decade of life. No doubt environmental influences in addition to hereditary influences are of importance in promoting the development of asthenia. Town life certainly favours slenderness of build; and so does much sitting during the years of growth—a practice inseparable from our type of culture. Still, we must not overlook the fact that hereditary weakness of the spine and an undue constitutional liability to fatigue are often associated with a disinclination for bodily exertion. Thus either element in the causation of asthenia reinforces the other. We cannot, however, agree that, as T. Brugsch[2] and other authorities declare, a marked constitutional tendency towards asthenia can be fully compensated by nothing more than carefully chosen bodily exercises during the years of youth.

Some have declared that disturbances in the functioning of the endocrine glands (the glands whose "internal secretions" supply such important constituents to the juices of the body) can account for the origination of asthenia. The fact is, however, that endocrine disturbances produce very definite changes which differ from those characteristic of the asthenic constitution. For a time it was the fashion to refer every constitutional anomaly to disturbances of internal secretion, and after a while a "polyglandular theory" replaced a "uniglandular theory", it being now supposed that many or all the glands of internal secretion were concerned in the production of asthenia. These glands may play a certain part in the matter, but for that very reason we are compelled to look elsewhere for the decisive cause of asthenia. The fact is that the functioning of the endocrine glands is itself dependent upon the hereditary equipment, and not that the hereditary equipment depends upon the endocrine glands.

B. Stiller,[3] who was the first to give an exhaustive account of asthenia, regarded it as a peculiar hereditary disease. Critics have objected that a constitutional anomaly is not in itself a disease. But, in its severer forms, "Stiller's disease" certainly comes within the cagetory of diseases as defined in the foregoing subsection "General Considerations". It is a condition which lies on the border line of the capacity for adaptation, one which causes many subjective disorders in those affected with it, inducing in them numerous abnormal reactions. Those who use the term "illness" or "disease" are too apt to think only of the infectious diseases and other maladies mainly due to environmental causes (see above p. 217). Still, the lesser degrees of asthenia must be regarded rather as constitutional anomalies than as constitutional diseases.

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[1] A. Wetzel, Die Stillersche Konstitutionsanomalie im Säuglingsalter, "Münch. Med. Wochenschrift", no. 35, 1922.

[2] Allgemeine Prognostik, second edition, 1922.

[3] Die asthenische Konstitution, Enke, Stuttgart, 1907.

Asthenics may to some extent be regarded as constituting a morbid race; and when we speak of an asthenic "race" the main difference in the use of the term, as contrasted with our usage when we ordinarily speak of the races into which the human species is divided, is that these latter are distinguished one from another by very numerous genes, whereas asthenics are distinguished from the rest of the population amid which they live by a few characters only. Some observers have tried to show that asthenia is definitely related to this or that of the great races of mankind, and some say that asthenia is a peculiarity of the slenderly built Nordic race. This is certainly wrong-headed. In each race, the ratio between chest measurement and stature has a significance peculiar to that race. A Mongol as slender as a Negro would be regarded as asthenic, i.e. morbid; whereas a Negro having the build characteristic of his race is perfectly healthy and possessed of full functional capacity. The distinctions between the great races of man can only be connected with asthenia to this extent, in that it seems possible that among the cross-breeds formed by the mingling of different races, in F_2 and the subsequent generations there may arise types in which an asthenic build and a weakness of body are associated.

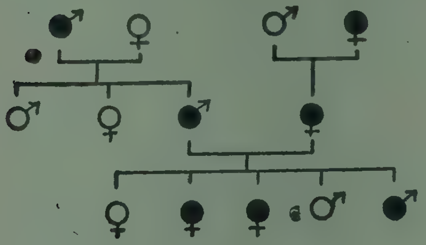

FIG. 84.
Habitus asthenicus, after Paulsen.

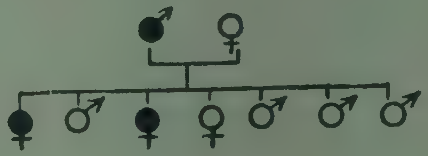

FIG. 85.
A family, three members of which were of the asthenic type, whereas all the others were of the pyknic type. After Paulsen.

Of course there are numerous transitions between a markedly asthenic habit of body and a normal habit of body, and between this normal habit and a markedly pyknic type. So numerous, indeed, are these transitional forms, that it is almost impossible to trace the heredity of constitutional types with clearness and precision. Although there may be good reason to suppose that certain abnormal heredity factors determine the asthenic habit, and that the heredity of these factors is simply dominant, the transitional bodily types, on the other hand, are to a considerable extent polymerically determined. This may be inferred from the study of 506 families carried out by C. B. Davenport.[1]

Akin to asthenia is *infantilism*, a constitutional anomaly characterised by an arrest of development at an immature stage. It is impossible to draw a sharp line between infantilism and asthenia. Indeed, many physicians are inclined to use the composite term "asthenic infantilism". In infantile human beings, the reproductive organs remain small and underdeveloped. Next to gonorrhoea, infantilism is probably the commonest cause of sterility in women. Should, however, conception occur in a woman whose uterus is infantile, abortion will probably ensue, for the fertilised ovum cannot implant itself successfully in the immature maternal organ. Infantilism also discloses itself in the general aspect of one who is affected by this condition. A young woman of 25 or 30 will, if infantile, have the features of a child, and will thus exhibit a spurious appearance of extreme youth. The mental condition of infantile persons likewise remains that of a child, so that they are unduly influenced by the immediate impressions of the senses and by the experiences of the moment, and can too readily be subjected to another's will. They are lacking in energy, show little capacity for steady work, and are fond of childish games.

Most cases of infantilism are unquestionably due in the main to hereditary influences.[2] On the other hand, environmental noxious influences (congenital syphilis, for instance) can inhibit development so powerfully that permanent infantilism may result. Often an infantile or hypoplastic constitution may be associated with tuberculosis. This association might be explained on the theory that tuberculosis in children can induce infantilism; but it seems to me a more probable explanation that infantile persons are exceptionally liable to tuberculosis. I think the latter view is confirmed by J. Bartel's observations.[1]

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[1] Body-Build and its Inheritance, Carnegie Institution of Washington, 1923.

[2] Borchardt, Ueber Abgrenzung und Entstehungsursachen des Infantilismus, "Deutsches Archiv für Klinische Medizin", vol. CXXXVIII.

Allied to asthenia and to infantilism is the condition known as *hypoplasia*, which manifests itself as weakness and underdevelopment of various organs. Persons affected with this condition usually succumb in infancy or early childhood; but some of them, suffering from the slighter degrees, may succeed in growing up. We are certainly justified in assuming that in many cases hereditary determinism is of decisive importance in originating the *hypoplastic constitution*. Still, we must bear in mind the possibility that noxious influences during infancy, and perhaps also environmental influences acting on the embryo within the mother's womb, may inhibit development to a degree which cannot afterwards be made good.

The notion of a "status degenerativus" seems to me unworkable, though it is one which a good many authorities favour. J. Bauer[2] defines this condition as "that most generalised form of constitutional anomaly in which the person affected by it manifests a larger or smaller number of the so-called signs of degeneration". The subdivisions of this degenerative state are said to be the "status lymphaticus, hypoplasticus, asthenicus, neuropathicus, exsudativus, arthriticus", and so on, and so on. Evidently, however, so vague a concept, embracing as it does the most diversified anomalies, is not available for the delimitation of a sharply defined condition.

I do not mean to imply that there is not a modicum of truth in the doctrine of the "stigmata of degeneration" or "signs of degeneration" which has developed out of the theory of degeneration formulated by the French alienist B. A. Morel.[3] To-day, indeed, we understand by this term degeneration, not an inescapable progressive doom, but the origination and expansion of morbific heredity factors. Thus the problem of the "stigmata of degeneration" is reduced to an attempt to discover whether there are external bodily characters justifying the inference that morbific heredity factors have been or are at work. Now there can be no doubt, in the light of our present knowledge, that a good many such characters can actually be recognised. When the chest measurement is too small, we infer that there is hereditarily determined bodily weakness; but, on the other hand, a large chest measurement does not necessarily mean that hereditary disease need not enter into our consideration, for excessive girth of the thorax may actually be the outcome of such disease (as in cases of pulmonary emphysema or of obesity). When the head is far too small (microcephaly), we know that there is feeble-mindedness; but excessive size of the head may also be the expression of a morbid condition (hydrocephalus). The unduly small circumference of the thorax or the head is the direct result of an under-development of the organ concerned; but when the circumference is unduly large, the causal sequence is a less obvious one. Furthermore, an obviously abnormal character which is *per se* devoid of morbid significance may be dependent upon the same heredity factor as an organic defect which is perhaps far less easy to recognise. Thus there is a *hereditary brittleness of the bones* (*osteopsathyrosis*) which is associated with a bluish-grey tint of the sclerotic (the "white" of the eye). In some of the cases there are also disturbances of hearing which are of the nature of otosclerosis. Several families have been studied in which the heredity of these conjoined affections was dominant.[1] In this case the bluish-grey colour of the white of the eye is a sign of degeneration which almost certainly indicates the existence of fragilitas ossium, and, with a moderate likelihood, that of otosclerosis. In addition, certain hereditary anomalies which are not dependent on the same gene are more frequently conjoined than the ordinary law of probability would lead us to expect. Thus according to Nettleship about 4 % of all deaf-mutes are affected with retinal atrophy (retinitis pigmentosa), and at least 3·3 % of persons suffering from retinal atrophy are deaf-mutes, although the normal frequency of each of these troubles is less than 1 per 1,000. The explanation is that deaf-mutism and retinitis pigmentosa are both of them recessive maladies which are especially apt to result from consanguineous marriage. Consequently, retinal atrophy is to some extent a "sign of degeneration" in relation to deaf-mutism; and conversely. An additional cause of the heaping-up of various hereditary defects is that persons with bodily or mental anomalies are shunned as partners by normal persons, and will therefore be especially prone to marry other defectives. That is why the "stigmata of degeneration" accumulate in certain families, and we interpret the occurrence falsely if we suppose that it points to the existence of a unified process of "degeneration". Degeneration is not a unified process. The truth is, rather, that the concept of degeneration is a composite one—is a name to denote the origination and the diffusion of the most multifarious morbid taints. T. Brugsch gives a list of what are commonly regarded as "signs of degeneration".[1]

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[1] Status thymico-lymphaticus und Status hypoplasticus, Deuticke, Leipzig and Vienna, 1912.

[2] Der Status degenerativus, "Wiener klinische Wochenschrift", no. 42, 1924.

[3] Traité des dégénérescences physiques, intellectuelles et morales de l'espèce humaine, Paris, 1857.

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[1] Conard and Davenport, Hereditary Fragility of Bone, "Eugenics Record Office Bulletin", no. 14, New York, 1915.

Funnel-breast is a malformation in which the lower end of the sternum is deeply depressed, so that there is a funnel-shaped hollow in this region. Families have been described, by J. Paulsen[2] and A. Peiper,[3] in which the heredity of funnel-breast was dominant. Sometimes, however, a generation was skipped. Apparently occupational pressure may produce a similar thorax ("cobbler's breast"), but Peiper is inclined to doubt whether there really is such a thing as cobbler's breast.

Lateral curvature of the spine (*scoliosis*) has been observed to run in families for several generations. There would appear to be dominant heredity factors at work. In former days the usual belief was that scoliosis was in some cases due to long-continued sitting in a crooked position, especially at school, and was in other cases an outcome of rickets. According to Schlesinger,[4] however, the school cannot really be blamed for the causation of lateral curvature. Probably, too, the importance of rickets in this connexion has been much overrated. It is certainly a remarkable fact that what is called "rachitic scoliosis" should seem to run in families without necessarily

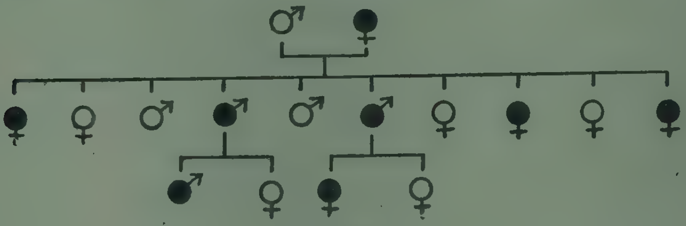

FIG. 86.
Funnel-breast, after Paulsen.

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[1] Allgemeine Prognostik, second edition, Berlin and Vienna, 1922, pp. 242 et seq.

[2] Ueber die Erblichkeit von Thoraxanomalien, "Archiv für Rassenbiologie", vol. XIII, no. 1, 1918.

[3] Ueber die Erblichkeit der Trichterbrust, "Klinische Wochenschrift", vol. I, no. 33, 1922.

[4] Die rachitischen und konstitutionellen Verbiegungen der Wirbelsäule, etc., "Archiv für Kinderheilkunde", vol. LXVIII, nos. 1 and 2, 1921.

being accompanied by any marked prevalence of the ordinary signs of rickets in members of these same families. Two illustrative genealogical trees have been published by H. Ziesch.[1] A family in which seven persons were affected with lateral curvature of the spine has been described by K. Classen.[2] Often lateral curvature is associated with a backward curvature of the spine (kyphosis).

Kyphosis itself, the "*round back*" of popular parlance, also has a marked tendency to run in families.[3] Dominant heredity factors are probably concerned in its production. Paulsen has published some family trees.

In the origin of *varicose veins*, prolonged standing and other conditions tending to interfere with the free return of venous blood to the heart play a considerable role, but we have to remember that in most persons such influences do not produce varicosities. The way in which the trouble runs in families shows that heredity must be a contributory cause. *Haemorrhoids* (*piles*), enlarged veins at the lower outlet of the intestine, are a special kind of varices, whose onset is perhaps favoured by a sedentary life, and is certainly favoured by pregnancy. But the influence of heredity must not be overlooked. M. J. Gutmann[4] has reported on a family in which fourteen members suffered from haemorrhoids, although few of these persons led sedentary lives.

Hanhart believes that there are heredity factors causing a general weakness of the connective tissue, which may manifest itself in various ways, such as hyperextensibility of the joints, splanchnoptosis (prolapse or falling-down of the viscera), flat-foot, rupture, varices, etc. Indisputably, such troubles are often associated. K. H. Bauer[1] likewise considers that the foundation of asthenia must be "a weakness and insufficiency of the connective and supporting tissues in general".

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[1] Statistisch-genealogische Untersuchungen über die Ursachen der Rachitis, "Archiv für Rassenbiologie", vol. XVII, no. 1, 1925.

[2] Vererbung von Krankheiten und Krankheitsanlagen, "Archiv für Rassenbiologie", vol. XIII, no. 1, 1918.

[3] "Lateral curvature" and "backward curvature" must be carefully distinguished from "angular curvature" of the spine. The last-named, also known as "Potts' disease", is due to tubercular destruction of parts of certain vertebrae, and is in quite a different category from the two others—though the term "hump-back" or "hunchback" is in folk-usage applied to any extreme manifestation of scoliosis, kyphosis, or angular curvature.—TRANSLATORS' NOTE.

[4] Zur Vererbung der Hämorrhoiden, "Archiv für Rassenbiologie", vol. XVII, no. 3, 1925.

In *chondrodystrophia* (also termed *achondroplasia* or *achondroplasty*) the limbs are dwarfed (micromelus or micromelia), whereas the head and the trunk are of approximately normal size. The malformation arises from an inadequate formation of bone by the epiphysial discs, upon the activity of which the normal growth in the length of the long bones depends. Chondrodystrophia varies in severity, and slighter forms of it are sometimes distinguished as chondrohypoplasia. Brachyphalangy, in which the finger-joints are rather too short, and which is apt to be accompanied by a moderate abnormal shortness of the limbs as a whole, may be regarded as a very mild manifestation of chondrodystrophia. H. Rieschbieth and A. Barrington have published 75 genealogical trees illustrating the family incidence of chondrodystrophia.[2] Some of these (Hergott's, for instance) show a dominant heredity, but dominance is not the rule in this affection. Indeed, it is especially the severer forms of chondrodystrophia which seem to depend on recessive heredity factors. A woman with extreme chondrodystrophia has so small a pelvis that delivery of a living child by the natural outlet is impossible. The obvious

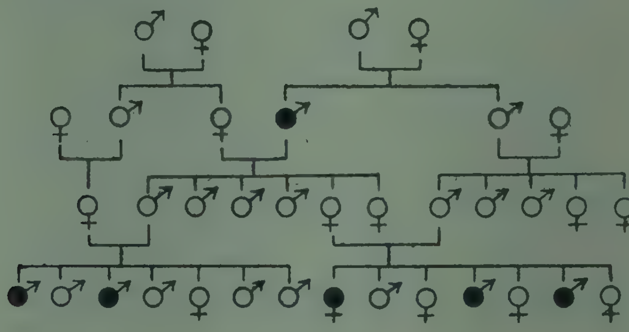

FIG. 87.
Chondrodystrophia, after Bonnevie.

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[1] Erbkonstitutionelle Systemerkrankungen und Mesenchym, "Klinische Wochenschrift", vol. II, no. 14, 1923.

[2] Dwarfism, in the Treasury of Human Inheritance, parts VII and VIII, 1912.

result will be (except in cases where Caesarean section is practised) the speedy elimination of dominant types in which such severe chondrodystrophia prevails. Kristine Bonnevie has published a very instructive family tree showing the obviously recessive incidence of chondrodystrophia.[1] Most of the other genealogical trees that have been compiled to illustrate the heredity of this malady are compatible with the idea that it is recessive.

In dogs, moderate chondrodystrophia is deliberately fostered in the case of dachshunds. Originally this type was favoured by breeders because such short-legged dogs were better able to enter fox-earths and badger-earths. The purposive selection of chondrodystrophic animals has produced a race endowed with this special quality.

Under the name of *dwarfism* there have been lumped together a number of conditions whose only common element is that the stature is abnormally small. Martin proposes to class as a dwarf any adult male human being whose stature is below 130 cm. (approximately 4 ft. 3 in.) and any adult female whose stature is below 121 cm. (approximately 3 ft. 11½ in.). One variety of dwarfism is represented by the before-mentioned chondrodystrophia. A second variety is constituted by what von Hausemann has called "primordial dwarfism" in which from birth onwards the entire frame is abnormally small. Dwarfs belonging to this category are when adult of like proportions with normal men, the only difference being that they are constructed on a smaller scale.

The Pygmy races of Africa and the eastern archipelagos are not essentially different from the "primordial dwarfs". In Pygmies, however, dwarfism is not to be regarded as morbid, being a selective adaptation to exiguous conditions of life.

In dogs, primordial dwarf types are selected by fanciers to produce special races.

Another kind of hereditary dwarfism is *infantile dwarfism*, in which the skull and the whole skeleton preserve infantile forms on into old age. The epiphysial discs of cartilage do not undergo ossification, and the reproductive organs remain like those of a child—but it is a mistake to regard the under-development of the gonads (reproductive glands) as the "cause" of the dwarfism. The condition of all the endocrine glands, and not that of the gonads alone, is determined by the hereditary equipment, and the under-development of all these glands of internal secretion together with the insufficiency of hormonal functioning is to be regarded as no more than a part of the general under-development. Infantile dwarfing, therefore, may be looked upon as an extreme manifestation of infantilism. A. Kraft[1] observed three infantile dwarfs in a kinship as the offspring of two consanguineous marriages. This speaks strongly in favour of a recessive heredity.

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[1] Arvelighetsundersokelser i Norge, Oslo, Christiania, 1915.

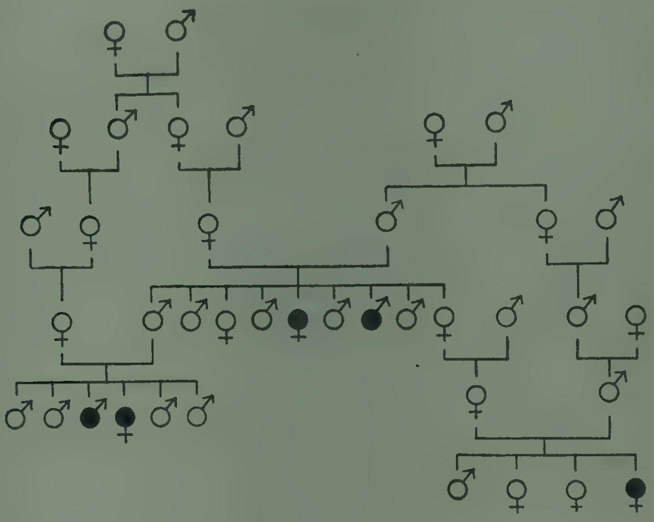

FIG. 88.
Dwarfism, after Hanhart.[2] (Extract.)

Ernst Hanhart, the Zurich geneticist, has thrown light on the heredity of hereditary dwarfism.3

He describes the hereditary dwarfism that prevails at Oberegg in the canton of Appenzell. "No one who visits this region can fail to be struck by the prevalence of well-proportioned and seemingly intelligent dwarfs, some of whom are by no means inclined to keep out of the way, being ready enough to turn an honest penny by the sale of picture postcards to visitors or by acting as servants in an inn. The dwarfs range in height from 85 cm. to 106 cm., but their parents and their brothers and sisters are persons of normal stature. We are told that the dwarfs were of normal size at birth. For instance, a female dwarf now 22 years of age and only 105 cm. in height is reported to have weighed 8 lb. when born. In the case of all of them, development is said to have gone on normally for a considerable time, the arrest of growth only beginning in the third year of life."

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[1] Ein Beitrag zum Erbgang des Zwergwuchses (Nanosomia infantilis), "Münchener Medizinische Wochenschrift", no. 24, 1924.

[2] Ueber heredodegenerativen Zwergwuchs, "Archiv der Julius-Klaus-Stiftung", vol. I, no. 2, Zurich, 1925.

3 Ueber die Bedeutung der Erforschung von Inzuchtsgebieten, etc., "Schweizerische Medizinische Wochenschrift", no. 50, 1924.

Hanhart found the same kind of dwarfism in the Samnauntal in the Lower Engadine; and also in the island of Veglia on the Dalmatian coast near Fiume, where it was erroneously regarded as "maritime cretinism". The heredity of the Hanhart type of dwarfism is manifestly recessive (see Fig. 88), and that is why it is encountered in isolated regions where inbreeding prevails.

The Hanhart type of dwarfism used to be called "pituitary dwarfism", when it was supposed to be an outcome of a disorder of the function of the pituitary body (a ductless gland situated at the base of the brain). Experiments on animals have in fact shown that removal of this organ is followed by an arrest of growth, but there seems to be considerable doubt whether Hanhart's dwarfism is due to lack of the pituitary incretion (hypopituitarism). Moreover, the term "heredodegenerative dwarfing" is a misnomer when applied exclusively to this error of development, seeing that all morbific heredity factors might be regarded as expressions of degeneration; and, on the other hand, this kind of dwarfing is not degenerative in the sense of being itself morbific.

Rieschbieth and Barrington (*op. cit.*) have published a number of genealogical trees showing the family incidence of dwarfism, the cases being classed under the general name of ateleiosis (a word coined from the Greek to signify that the goal of development has not been reached). These writers fail, however, to distinguish adequately between the various forms of dwarfism. Three genealogical trees published by Gilford and Levy to illustrate the family incidence of what is presumed to be primordial dwarfism, show a dominant or an intermediate heredity; on the other hand a genealogical tree published by Taruffi suggests a recessive heredity. In one family a dwarf father and a dwarf mother had 14 children of normal stature. We may assume that here the dwarfing was due to a different kind of heredity factor in the respective parents, and that, the taint being recessive in both instances, these factors were masked in each case by an allelomorphic normal factor derived from the other parent.

G. Selle has reported on a family in which a moderate degree of dwarfism showed a dominant heredity.[1] Speaking generally, however, we find that, in dwarfism, recessive heredity is much commoner than dominant.

Apart from these rare morbific heredity factors, there are of course very many normal heredity factors which influence the stature. The differences which come within the range of the normal as far as this character is concerned must, therefore, be regarded as polymeric in their heredity.[2]

Among the endocrine glands, the pituitary has the most marked effect upon the stature. When the pituitary incretion is excessive in quantity (hyperpituitarism) a peculiar form of gigantism results, the overgrowth affecting especially such distal parts as the hands, the feet, the nose, and the chin. Thus arises the condition known as *acromegaly*, which is, according to L. R. Grote,[3] often hereditary, so that we must suppose a hereditarily transmissible hyperfunctioning of the pituitary to be at work. Presumably there is also a hereditary hypopituitarism attended by arrest of bodily growth.

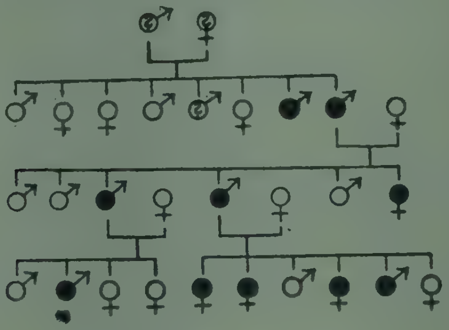

FIG. 89.
Dwarfism, after Selle.

The thyroid, too, has a marked influence upon growth. In *cretinism*, whether endemic or sporadic, the root cause of the trouble is hypothyroidism, as a result of which growth does not take place as it should, so that in goitrous regions cretinism is the main cause of dwarfism. On the other hand, persons affected with hyperthyroidism are usually taller than the average. Characteristic of cretinism is a depression of the root of the nose, which results from a lack of sufficient growth in antero-posterior length at the base of the skull. A similar condition of the nose is manifest in other cases where growth in the cartilaginous zones of the bones is insufficient, as for instance in chondrodystrophia. Finally, in this connexion, it should be mentioned that the reproductive glands have considerable influence upon the stature.

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[1] Ueber Vererbung des echten Zwergwuchses, Jena, 19

[2] C. B. Davenport, Inheritance of Stature, "Eugenic ecord Office Bulletins", no. 18, 1917.

[3] Grundlagen ärztlicher Betrachtung, Springer, Berlin, 1.

Rickets, by making the bones bend, can reduce the stature a good deal. Nevertheless, as already said, the notion of "rachitic dwarfism" has been pushed much too far.

The *situs viscerum inversus* (*inverted viscera*), a rare condition in which the thoracic and abdominal viscera are laterally transposed (so that the heart points to the right instead of the left, and so on), has occasionally been observed in brothers and sisters. In a case described by K. Ochsenius, two brothers with inverted viscera were the children of cousins.[1]

H. W. Siemens' study of twins has shown that *lefthandedness* is not to so large extent the outcome of heredity as used to be supposed.[2] He examined 21 pairs of mon-oval twins in whom one member of the pair was righthanded and the other lefthanded; and Weitz found similar conditions in 7 pairs of twins among 18. It would seem to be very common in twins that one member of the pair should be righthanded and the other lefthanded. The general frequency of lefthandedness is said to be from 4 to 5 % in adults and at least 10 % in children, the difference being accounted for by the fact that so many lefthanded children are made righthanded by education. Thus we see that which hand is predominantly used depends to a considerable extent upon external influences.

As a supplement to the account of the anomalies of bodily form, we shall now consider to what extent hereditary predisposition plays a part in producing *twins*. Mon-oval (monozygotic) twins are formed by the complete severance of one fertilised ovum into two halves, each of which then develops into an independent child. When the severance is incomplete, so that the two children remain attached to one other by continuity of tissues or organs (a very rare occurrence), malformation is obvious. Di-oval (dizygotic) twins are formed out of two distinct ova which are simultaneously fertilised—or successively fertilised, the process being known as superfecundation when the two ova belong to the same menstrual period, and as superfoetation when they belong to different menstrual periods. Thus in the case of mon-oval twins the members of the pair will have identical hereditary equipments, whereas in the case of di-oval twins there will not as a rule be more similarity in this respect than happens in the case of ordinary brothers and sisters. Since mon-oval twins are always of the same sex, whilst among di-oval twins males and females occur in the usual proportions, it is easy to calculate the proportion of twins that are mon-oval, and the calculation shows that about one fourth of all pairs of twins must be mon-oval.[1] Thus, the ordinary proportion of twin births to other births being about 1 : 80, the proportion of mon-oval twin births will range from 1 : 300 to 1 : 350. Weinberg found that in Wurtemberg among the mothers of twins of different sexes the probability of their giving birth to twins was twice the average, and among the sisters of such mothers was actually thrice the average. It is obvious, therefore, that heredity must play a part in giving rise to the tendency to bear di-oval twins. The fact that the sisters of the mothers of twins show a greater proclivity than the mothers themselves indicates that some, at least, of the heredity factors at work must be recessive. Since only from 5 to 6 % of the mothers of twins bear twins a second time, we must recognise that only in exceptional instances does the tendency to bear twins become actual. Perhaps, then, environmental influences play a notable part in the production of twin births. Weinberg did not find that the tendency to produce mon-oval twins ran in families, but G. Dahlberg rightly points out that the data collected by Weinberg are not incompatible with such a possibility.[2] As the outcome of an exhaustive study of the peasant population of Norway, Bonnevie[3] arrives at the same results as Weinberg.

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[1] Ueber familiären Situs inversus, "Monatsschr. für Kinderheilkunde", vol. XIX, no. 1, 1921.

[2] Ueber Linkshändigkeit, "Virchows Archiv", vol. CCLII, no. 1, 1924.

In human beings the production of twins must be regarded as abnormal. From 5 to 6 % of twin pregnancies are characterised by the occurrence of the dangerous disease eclampsia (puerperal convulsions) in the mother.[1] Owing to the excessive distension of the uterus, one fourth of twin pregnancies end in premature delivery. Stillbirths and deaths in infancy are more than twice as common in twins as they are in ordinary children.

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[1] W. Weinberg, Die Anlage zum Mehrlingsgeburt beim Menschen, "Archiv für Rassenbiologie", vol. VI, nos. 3–5, 1909.

[2] Twin Births and Twins, Stockholm, 1926.

[3] K. Bonnevie and A. Sverdrup, Hereditary Predispositions to Dizygotic Twin-Births, "Journal of Genetics", vol. XVI, no. 2, 1926.

Numerous genealogical trees have been published showing that the tendency to give birth to twins runs in families. In view of the rarity of twin births, this accumulation in particular families cannot depend upon chance, and it cannot be accounted for as the outcome of the comparatively small effect (researches of Weinberg and of Bonnevie) of the recessive heredity factors of twin births. The cases in which a woman gives birth to twins several times in succession may be accounted for in the light of Bumm's observation[2] that in the ovaries of a woman who died when giving birth to twins, almost every Graafian follicle contained two ova. But this explanation does not apply to all the family trees that have been published to illustrate the occurrence of twin pregnancies. E. Dirksen[3] quotes an American author named Valenta as having reported: "A mother and a daughter, both epileptic, had 32 children each: the mother had 6 times given birth to twins, 4 times to triplets, and 2 times to quadruplets; the daughter, forty years of age at the time of writing, was then in a condition of multiple pregnancy, had 3 times given birth to twins, 6 times to triplets, and 2 times to quadruplets." In 1923, A. Peiper[4] published a genealogical tree which has been several times reprinted, and which I subjoin. At the same time I think it expedient to say that I find it simply impossible to believe in the accuracy of such a tree. It was obviously based on the woman's own statements, and one must suppose a hysterical

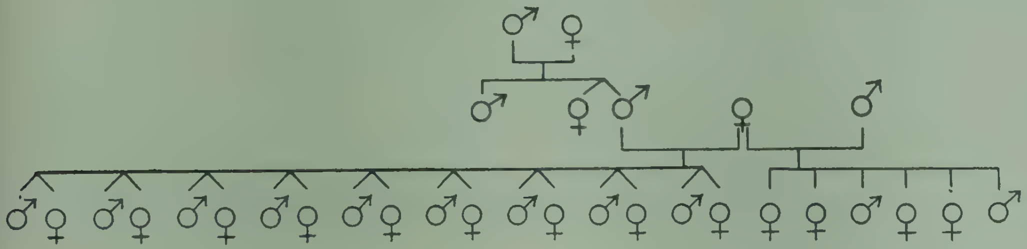

FIG. 90.

Ostensible hereditary transmission through the male of the tendency to produce twins, after Peiper.

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[1] F. Weber, Mehrfache Schwangerschaft, in Döderlein's Handbuch der Geburtshilfe, vol. III, 1920.

[2] Zentralblatt für Gynekologie, no. 19, 1923.

[3] Asoziale Familien, "Deutsche Zeitschrift für öffentliche Gesundheitspflege", vol. I, nos. 5 and 6, 1924, p. 104.

[4] Zur Vererbung der Zwillingsschwangerschaft durch den Mann, "Klinische Wochenschrift", vol. II, no. 35, 1923.

imagination to have been at work. Pregnancy, ordinary pregnancy, already plays a notable part in the fantasies of hysterically inclined women; and obviously twin pregnancy (to say nothing of triplets or quadruplets) would seem to such a woman an extremely "interesting condition". But to make herself appear interesting is the hysterical woman's outstanding desire! The family tree illustrating twin pregnancies published by Zondek[1] obviously relates to the same woman, although, be it noted, the data are not identical. Like criticism must be applied to some even more fabulous cases recorded in the earlier literature of the topic. The dubious instances I have actually reproduced were worth reproducing because of their important bearing upon the methodology of genetic research.

I print as Fig. 91 a trustworthy genealogical tree which E. Meirowsky was able to compile as the outcome of his study of twins.[2] The question whether the tendency to give birth to mon-oval twins is likewise a family proclivity, is one which must be decided by investigation among the kinships of mon-oval twins, concerning which ample data are now in course of collection.

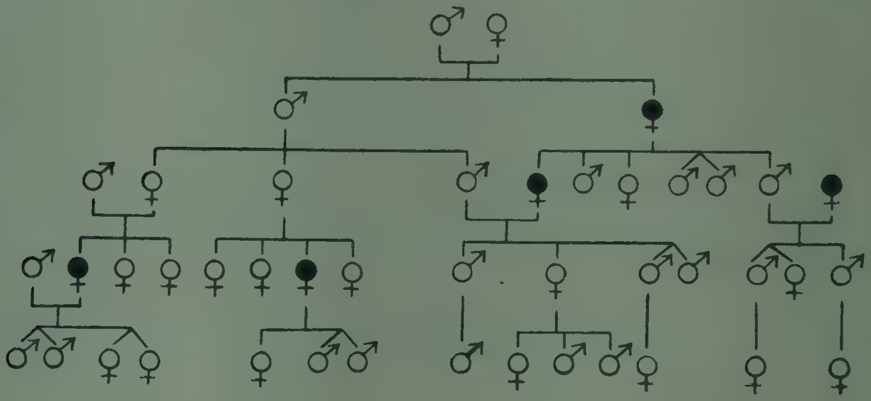

FIG. 91.
Twin pregnancy, after Meirowsky.

Of course the heredity factors leading to the production of mon-oval twins must be very different from those leading to the production of di-oval twins. The usual cause of the latter one may suppose to be the simultaneous release of two matured ova ready for fertilisation. The underlying, presumably recessive, heredity factor must be homozygotically present in the mother. In the case of mon-oval twins, on the other hand, one may assume the operative heredity factor to be one which determines an unduly complete division of the fertilised ovum. If we are concerned with recessive factors, the homozygotic coincidence of two such heredity factors would (we must suppose) only occur at the time of the fertilisation of the ovum out of which the mon-oval twins arise. Consequently, a hereditary predisposition to produce mon-oval twins could only become effective in every fourth pregnancy at most. It would, however, be exceedingly probable that even a homozygotic tendency of this kind would only become effective in a fraction of the theoretically possible instances—as manifestly happens in the case of di-oval twin pregnancies. These considerations will explain why it is so much more difficult to demonstrate the hereditary predisposition to mon-oval than that to di-oval twin pregnancies, even though the importance of heredity may be equal in the respective cases.

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[1] Ueber Zwillingsschwangerschaft, "Zentralblatt für Gynäkologie", no. 19, 1923.

[2] Kleinere Mitteilungen zur Erblichkeitslehre, "Archiv für Rassenbiologie", vol. XVII, no. 4, 1926.

If the tendency to produce di-oval twins displays itself through the simultaneous maturation of two ova, we should expect to find that the father counts for nothing in this matter. C. B. Davenport,[1] studying 644 births in families where twin pregnancies had occurred, found that the sisters of women who had given birth to twins procreated 5·5 % of twins, that the brothers of women who had given birth to twins procreated 4·5 % of twins, that the sisters of men whose wives had given birth to twins procreated 8·2 % of twins, and that the brothers of men whose wives had given birth to twins procreated 6·5 % of twins—whereas the average percentage of twin births is only 1·1. Davenport's material is too scanty to enable us to draw trustworthy inferences. If, however, further inquiry shows that the father really has an influence in this matter, we must conclude that the simultaneous maturation of two ova is not the decisive factor in the production of di-oval twins. An alternative possibility is that normally as soon as an ovum has been fertilised there are formed in the uterus substances which inhibit the fertilisation of any more ova. A hereditary inadequacy in the process by which such substances are formed might be of paternal just as well as of maternal origin.

6. HEREDITARY DIATHESES (SUSCEPTIBILITIES)

When we speak of diatheses we denote the existence of abormal susceptibility to certain environmental influences which normal persons can endure without ill effect. In many instances the susceptibility is so great that the ordinary influence of everyday life can give rise to morbid manifestations.

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[1] Influence of the Male in the Production of Human Twins, "American Naturalist", vol. XXXIV, 1920.

Diatheses which are as well marked as this make themselves obvious in all individuals affected by them. Slighter diatheses, on the other hand, may remain permanently hidden if the environmental conditions are persistently favourable.

Diatheses of all kinds are especially conspicuous during childhood, and for this reason paediatrists are peculiarly inclined to speak of diatheses. But diatheses are not limited to childhood, and not a few of them never show themselves until the subjects of them are well on in years. We shall begin, however, with the consideration of those which are mainly active in children.

There are, nevertheless, exceptional difficulties attendant on the study of the heredity of the diatheses of children. One of these difficulties is that many diatheses remain hidden when environmental conditions are favourable to the maintenance of health, and therefore unfavourable to the manifestation of the diatheses. Another difficulty is that parents, at the time when we approach this study of the diatheses of children, are naturally past the age when such diatheses are active, and grown-up persons' statements as to their health during childhood must usually be accepted with considerable reserve. Yet another difficulty is that the delimitation of diatheses and the distinction between them is hardly possible until their hereditary biological relations have been clearly ascertained. Prior to this, a classification can only be symptomatic, and consequently provisional.[1]

The best known of the diatheses of childhood is what is termed the *exudative* or *inflammatory diathesis*. Infants suffering from it have very sensitive skins, and are prone to itching and eruptive forms of dermatitis (strophulus, prurigo, etc.). The previously described constitutional eczema of nurslings comes within this category. Crusts are apt to form on the hairy scalp ("scald-head") and upon the cheeks (impetigo, etc.). The mucous membranes, too, are abnormally sensitive ("geographical tongue"). Such children are much inclined to catarrhal and asthmatic troubles.

Many doctors include various other manifestations (disorders of nutrition, swellings of the lymphatic glands, abnormalities of behaviour) among the expressions of the exudative diathesis. Since Pfaundler could not find that such conditions had any regular connexion with the diathesis in question, I have thought it better to restrict myself to a mention of the symptoms of the inflammatory diathesis in the narrower sense of the term.

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[1] Cf. M. von Pfaundler, Ueber Wesen und Behandlung der Diathesen im Kindesalter, Bergmann, Wiesbaden, 1911.

The inflammatory diathesis is beyond question in the main a product of hereditary predisposition. The way in which it tends to run in families suggests a dominant heredity, but there are other possibilities. According to Czerny and Pfaundler, the taint is more often derived from the mother than from the father. Pfaundler reports that female transmitters of the heredity factors may themselves be apparently free from the diathesis. Thus a notable part of the heredity factors of the exudative diathesis would seem to be recessively sex-linked. This harmonises with the experience that many more boys than girls suffer from it. In 200 cases, Pfaundler found a proportion of 2 boys : 1 girl.

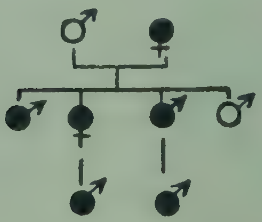

FIG. 92.
Disposition to dermatitis (eczema) in childhood, after Pfaundler.

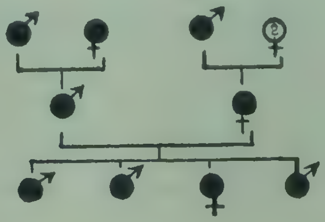

FIG. 93.
Disposition to catarrh of mucous membranes in childhood, after Pfaundler.

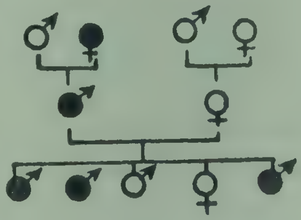

FIG. 94.
Dystrophic diathesis, after Pfaundler.

A second diathesis peculiar to children shows itself as an abnormal proneness to nutritive disturbances in infancy. "Infants with a favourable hereditary endowment often thrive on a diet which it gives a paediatrist the creeps to hear of", writes Pfaundler. Other infants will show a marked intolerance of non-human milk, such as cow's milk, and Pfaundler speaks of this peculiarity as *heterodystrophy*. In rare cases, on the other hand, we find that a nursling cannot thrive on its mother's milk.[1] The severe nutritive disorders of infancy are to a greater

[1] The trouble in such cases is often a result of unwise feeding on the

extent the outcome of the *dystrophic diathesis* than of injudicious feeding. At any rate this applies to some of those conditions in which the nursling's nutrition completely breaks down, and which used in former days to be described by some such vague term as "atrophy" or "marasmus". In the same category we may include the tendency to marked variations in the water-content of the body with consequent fluctuations in weight ("tropholability"). J. K. Friedjung[1] found that the parents of infants which suffered from nutritive disturbances notwithstanding all possible care were themselves usually prone to digestive disorders, whereas the parents of children that throve at the breast were usually persons with a vigorous digestion. These facts suggest that dominant genes play a considerable part in causing the dystrophic diathesis.

Very notable is the way in which mon-oval twins tend to thrive, or otherwise, together in this matter of nutrition. Both members of the pair usually have the same weight-curve. Arrest of increase or actual decline will occur simultaneously in both.[2] In the case of di-oval twins there is far less harmony in this matter, which shows that identity of treatment is not the main cause.

Thirdly we have to consider what is known as the *lymphatic diathesis*, which manifests itself in childhood by excessively large tonsils, swelling of the cervical lymphatic glands, and overgrowth of other lymphatic organs (status lymphaticus). Children thus affected, are usually inclined to suffer from catarrh of mucous membranes and from tonsillitis, and in consequence of the overloading of their tissues with water they have a pasty look. When children with a lymphatic constitution become tubercular, the tuberculosis is usually of the type which in former days was called scrofula (and earlier still, king's evil)—tubercular disease of lymphatic glands and of bone with "cold

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nursing mother's own part, and especially of a diet too rich in fats and proteids.

[1] Ernährungsstörungen der Brustkinder und Konstitution. "Zeitschrift für Kinderheilkunde", 1913.

[2] F. Rohr, Ueber eineiige Zwillinge, "Zeitschrift für Kinderheilkunde", vol. XXVI, 1920, p. 304.—Ibid., Ernährungsstörung gleichartigen Verlaufs bei eineiigen Zwillingen, "Deutsche Medizinische Wochenschrift", no. 28, 1923.

abscesses". The lymphatic diathesis obviously depends in great measure on the working of dominant heredity factors.

It used to be supposed that one result of the lymphatic diathesis was an abnormal persistence of the thymus gland beyond the years of childhood ("status thymico-lymphaticus"), and that this accounted for deaths occurring in the course of slight operations or for some other seemingly inadequate cause. Mental anomalies and suicidal tendencies were also attributed to such a persistence of the thymus. The theory has been discredited by recent observations. Though it is true that in young persons who die suddenly the thymus is usually in a fine state of preservation, whereas this is not the case in persons of the same age who die after a long illness, the difference seems to depend solely on the fact that the thymus wastes more than most organs during prolonged illness.

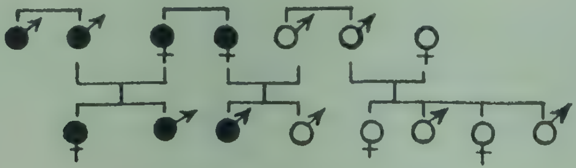

FIG. 95.
Status lymphaticus, after Pfaundler.

In some sickly children enlargement of the lymphoid tissues of the pharynx, and especially enlargement of the tonsils, are conspicuous features of their morbid condition, and then clinicians speak of an *adenoid constitution.* In the post-nasal region there is a mass of lymphoid tissue sometimes called the mid-pharyngeal tonsil or Luschka's tonsil. Enlargement here ("adenoids") interferes with nose-breathing, and entails various disorders, such as deafness and arrest of mental development. Children suffering from adenoids breathe mainly

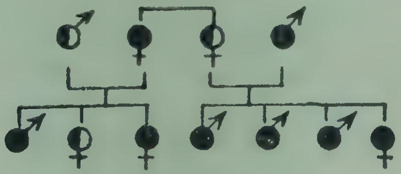

FIG. 96.
Status lymphaticus (◑) and disposition to catarrh of mucous membranes (◐), after Pfaundler.

through the mouth, and this is supposed to make the upper jawbones narrow and the palatal arch narrow and pointed. To some extent, however, the connexion may be differently interpreted. We may well suppose the narrowness of the palate to be a direct expression of the hereditarily determined narrowness of the upper jawbones upon which the narrowness of the nasopharyngeal space likewise depends.

The adenoid constitution is very common. According to A. Peyser,[1] among 800,000 Dutch schoolchildren there were 6 % with obvious morbid manifestations resulting from enlargement of the tonsils. The adenoid constitution seems to be especially frequent in the population of north-western Europe and in that of the United States (so largely peopled by immigrants from north-western Europe). Certainly these stocks are prone to have the narrow noses which are apt to make the respiration less free than it should be. Among about 1000 especially gifted children concerning whom Terman made enquiries in California, approximately half had had adenoids removed. We are not, however, justified in inferring from this, that talent is in some way linked up with the adenoid constitution.

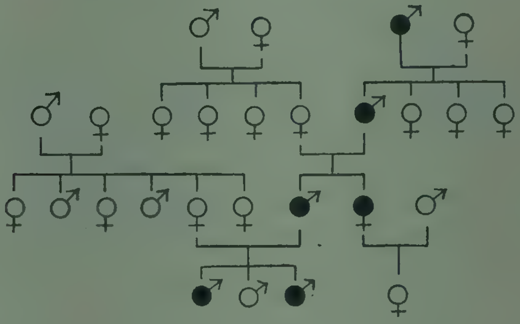

FIG. 97.
The adenoid constitution (personal observation).

In their investigations concerning twins, Siemens and Weitz found that mon-oval twins were always closely alike in respect of the size of the tonsils, the existence of adenoids, and so on. My own experience enables me to publish here a genealogical tree showing the family incidence of the adenoid constitution for four generations. The heredity was apparently dominant.

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[1] Hals-, Nasen-, Ohrenleiden, in Grotjahn's Soziale Pathologie, second edition, Springer, Berlin, 1923.

I do not consider that we have sufficient ground for speaking of a "vagotonic" or "eosinophil" diathesis. Asthma and certain other morbid states which have been classed under this head will come up for discussion later. Nor shall I describe a neuropathic or psychopathic diathesis, for psychopathies will be considered in a different connexion. As to the alleged "arthritic diathesis", which is supposed to be responsible for various other troubles in addition to affections of the joints, a number of morbid states having no real interconnexion have obviously been subsumed under this name.

Morbid states characteristic of the inflammatory, the dystrophic, the lymphatic, and the neuropathic diathesis, respectively, occur more frequently in association than the frequency of the several conditions per se would lead us to expect. Thus in some clinical material which came under the observation of M. von Pfaundler,[1] among 55 lymphatic children 44, that is to say about 80 %, also had inflammatory conditions, and 25, approximately 45 %, also had psychopathic conditions. By such experiences many physicians have been led to extend the concept of the "exudative diathesis" too widely, so as to include in it various other diathetic conditions in addition to inflammatory. Pfaundler has, however, shown that such "combined diatheses" can be analysed into a number of distinct diatheses which combine one with another in accordance with the law of probability, that is to say as independent genes. The unduly frequent concomitance of several of these morbid states will thus be obviously explicable on the hypothesis that certain heredity factors can cause several such conditions but others only one. Pfaundler reminds us that other hereditary characters, such as the colour of hair, eyes, and skin, are correlated, and yet not absolutely so. This comparison seems to hit the mark. We know that certain heredity factors can simultaneously determine the tint of hair, eyes, and skin; that others can influence the tint of hair and eyes only, of eyes and skin only, or of hair and skin only; and that yet others can influence the tint of hair only, eyes only, or skin only. (See pp. 136 et seq.) The correlation of the various diatheses is probably to be explained in the same way.

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[1] Kindliche Krankheitsanlagen (Diathesen) und Wahrscheinlichkeitsrechnung, "Zeitschr. für Kinderheilkunde", 1912.

Again, when one heredity factor becomes active or manifest, this can favour the activity or manifestation of another heredity factor. For instance, inflammatory changes in the skin and the mucous membranes (itching eruptions, bronchial catarrh) can readily disturb the psyche and thus induce manifestations of the psychopathic diathesis which would otherwise have remained quiescent. Undue serous infiltration of the skin as as outcome of the dystrophic or the lymphatic diathesis will induce a liability to inflammatory changes in the skin which has a very different significance from a primary disposition to skin eruptions. Finally the circumstance that persons predisposed to disease are more apt to marry persons likewise predisposed than to marry healthy individuals, leads to a veritable heaping up of diatheses in certain families and in certain patients.

When different heredity factors are able to produce clinical pictures which resemble one another in manifold ways, their study becomes very difficult. Observations made on one family alone will rarely suffice to elucidate the working of a morbid heredity; and when we aggregate the observations made upon the members of several families, there is always a risk that we may be lumping together things which really belong to different categories. An additional difficulty is that genuinely identical clinical results may arise out of the working of heredity factors that are essentially distinct in their method and course of operation. For instance, the inflammatory reactions which occur in persons suffering from the inflammatory diathesis do not occur in such individuals only nor exclusively as a result of a morbific hereditary equipment, for they may ensue also in persons with a "normal" hereditary equipment—though not, as in the subjects of the inflammatory diathesis, as the outcome of ordinary environmental conditions acting with an ordinary intensity, but only when the conditions are peculiarly unfavourable and when the stimuli able to induce an inflammatory reaction are exceptionally powerful. I think it not only possible but probable that there are certain heredity factors which work as activators or sensitisers in such a way that stimuli which in the absence of these factors would be ineffectual are able when they are present to give rise to inflammatory reactions. If this be so, then for practical purposes such a factor would be a factor of the inflammatory diathesis. If we suppose a case in which the presumed intensification of stimuli would occur only when two cooperating factors of like kind were at work, then we should have to do with recessive heredity factors of the inflammatory diathesis; but if one factor acting unaided were competent to produce the kind of effect we are considering, then it would have to be looked upon as a dominant heredity factor of the inflammatory diathesis. But the possibility of the occurrence of inflammatory reactions in persons whose hereditary equipment was normal would still remain—whenever the external noxious influences were peculiarly virulent. We see, therefore, that one and the same morbid condition may result, in one case from the working of dominant heredity factors, in another from the working of recessive heredity factors, and in a third simply and solely from the working of environmental causes. I think the same reasoning applies to many other morbid conditions besides those which are or may be classed as manifestations of the inflammatory diathesis.

Nay more, the possibility of inflammatory reaction which is part of the normal hereditary equipment is obviously favourable to survival, since it is part of the mechanism whereby we defend ourselves against noxious influences or relieve ourselves of their undesirable results. We must bear in mind that the absence or the enfeeblement of such reactions may itself be morbid, may be an expression of morbific heredity factors. Thus children which seem to thrive on a very unnatural diet, and those whose skins do not become inflamed when exposed to strong stimuli, are really the subjects of a morbid heredity. Presumably they lack potentialities for reaction which in other conditions might have been essential to survival. The response to stimulus should be neither too great nor too small, but should lie between these extremes.

The liability of little children to *rickets* (in Germany, often though foolishly called "the English disease") is likewise in great measure dependent upon hereditary predisposition, so that we have good reason for speaking of a *rachitic diathesis*. Rickets is characterised by a defective development of the bones; and even bones which have already been formed normally may be decalcified, being thus made soft and liable to abnormal bendings. This often leads to marked deformity of the bones of the legs, the pelvis, etc. The formation of the teeth is also interfered with in this disease.

It is true that the onset of rickets is greatly favoured by a lack of sunlight and exercise, and also by unsuitable feeding (excessive quantities of fat and the lack of certain accessory food factors); but Pfaundler has shown that the frequency and severity of the disease are mainly dependent upon hereditary predisposition. In certain families, although the children are well cared for, several or all of them suffer severely from rickets; and in other families, although the circumstances are most unfavourable, none of the children become affected with rickets. Siegert[1] has published family histories which are very instructive in this respect. Siegert and Elgood have also reported cases in which a woman, herself healthy and married to a healthy adult who had suffered severely from rickets in childhood, had several children which became severely affected with rickets; but in subsequent marriage to a man free from rickets, she gave birth to children which remained normal. Ziesch, too, has published a family tree of this kind in a work which has notably contributed to our knowledge of the hereditary character of rickets.

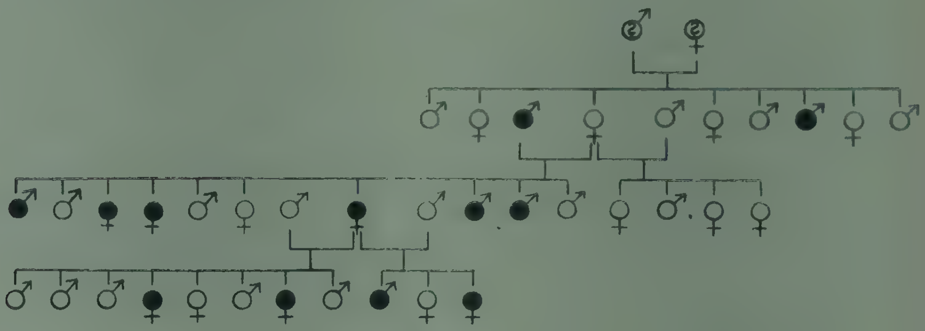

Fig. 98.
Rickets (after Ziesch).

The genealogical trees published by H. Ziesch[2] show, above all, that rickets is not a biological unity, as has been previously supposed; or, perhaps, that there are several specific hereditary tendencies which have hitherto been erroneously classed together as rickets. In many families in which rickets is hereditary, we find the heredity complicated in this way, that in one of the families a good many persons will suffer from curvature of the spine; in another, many of them will have pelvic deformities; in a third, there will be proneness to malformation of the skull; in a fourth, several of the members will be knock-kneed; and so on. I publish some of these family trees under other heads, for I regard their connexion with rickets as questionable.

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[1] Beitrag zur Lehre von der Rachitis: die Erblichkeit, "Jahrbuch für Kinderheilkunde", 1903.

[2] Statistisch-genealogische Untersuchungen über die Ursachen der Rachitis, "Archiv für Rassenbiologie", vol. XVII, 1925, no. 1.

Siemens and Weitz, in their study of mon-oval twins, found a marked similarity in respect of the incidence of rickets.

There is a certain correlation between the rachitic diathesis and the *spasmodic diathesis.* Most of the convulsive disorders of infancy ("fits", "tetany"), which the laity are fond of blaming upon the "teeth", are to be regarded as manifestations of the spasmodic diathesis; and the same is true of laryngismus stridulus (child-crowing). In many families we find that most of the children perish from one of these convulsive disorders. According to Thiemich and Birk,[1] when children affected with the spasmodic diathesis survive into adult life, they usually display various anomalies.

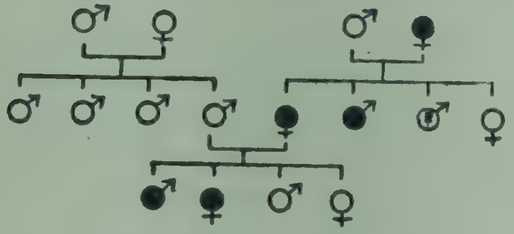

FIG. 99.
Hyperthyroidism (personal observation).

The tetany of adults, due to a defect of function in the parathyroids and the thyroid, has also, at times, been observed to run in families.[2]

Among the diatheses which do not manifest themselves until adult life has begun, *hyperthyroidism* plays a leading part. This displays itself as over-action of the thyroid, which results in an undue acceleration of the general nutrition, and when marked and acute is known as *exophthalmic goitre* (Graves' disease). In these patients there is usually an obvious enlargement of the thyroid, and during acute attacks of the disease the eyes may be extremely prominent (exophthalmos). There are, however, many persons suffering from hyperthyroidism in whom the course of the affliction is chronic, lasting in a subdued form for years and years without leading to the developed clinical picture of exophthalmic goitre. The usual symptoms of these subdued forms of hyperthyroidism are a persistent increase in the frequency of the pulse and a fine tremor of the fingers. Such persons may be said to be "out of breath" before they begin to run. They suffer from palpitation, unduly accelerated nutrition, have a warm, damp skin, are tremulous, and are prone to mental excitement. Their functional capacity is correspondingly reduced. Owing to the long-continued strain upon the heart, this organ undergoes enlargement, and ultimately it is apt to break down.

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[1] Ueber die Entwicklung eklamptischer Kinder in späterer Kindheit, "Jahrbuch für Kinderheilkunde", 1907.

[2] F. A. Kehrer, Zur Pathogenese der Tetanie, "Klinischc Wochenschrift", 1925, no. 40.

Typical Graves' disease is eleven times commoner in women than in men. It does not follow from this that the hereditary predisposition to the disorder is unequally distributed in the two sexes. We may suppose that the predisposition becomes active more frequently and more severely in women, just as non-hereditary goitre is much commoner and much larger in women than in men. Minor forms of hyperthyroidism are also fairly frequent in males. Not much is known as yet concerning the heredity of Graves' disease.[1] There is some reason to suppose that the heredity is dominant, but that for its active manifestation there must co-operate certain contributory external causes as to whose nature we have no certain information. Comprehensive family trees might throw light upon the matter, but unfortunately these are still lacking.

Sittmann reported that in the Baltic provinces, Graves' disease is found to run in families, but only in the dominant German caste. Here in Germany, however, the disease is commoner among Jews than among Gentiles.

The name *goitre* is a general term for all enlargements of the thyroid. In ordinary goitre, however, the activity of the thyroid, far from being increased as in hyperthyroidism, is usually diminished. Only in the slighter cases of goitre is there an unmistakable increase in the thyroid tissue proper. In severe cases of goitre, the enlargement mainly depends upon the deposit of colloid substance and upon the overgrowth of connective tissue. It has been usual to distinguish between "endemic goitre", which is common in the Alps and also in the mountainous regions of South Germany, and "sporadic goitre", which is occasionally observed in the plains and the coastal regions of North Germany. Goitre is endemic in some of the limestone regions of Great Britain. From its frequency in Derbyshire, it is sometimes spoken of as "Derbyshire neck". Goitre is commoner and more severe in women than in men.

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[1] E. Schultheiss, Ueber Erblichkeit des Morbus Basedowii, Jena, 1909 (a dissertation); E. Goldberg, Ueber die Erblichkeit der Basedowschen Krankheit, Berlin, 1910 (a dissertation).

In a family living in a region where goitre was not endemic, H. W. Siemens[1] was able to trace the occurrence of goitre for six generations. The taint seems to have been dominant. Only female members of the family were affected with the disease, and the inheritance could only be followed in the female line. In a family described by Agnes Bluhm,[2] the inheritance of goitre seems to have been dominant on the whole, and the affection was transmitted through healthy males as well as females.

There can be no doubt that endemic goitre (which is so common in certain regions that the majority of women have goitre) is mainly due to external noxious influences. The decisive factor would appear to be a lack of iodine in the food and in the environment generally. Iodine, though in very small quantities, is essential to the tissue changes of the human organism. Should the requisite minimum of iodine not be supplied in the drinking water and the food, there ensues in predisposed persons an enlargement of the thyroid—where the body iodine is normally stored. The first stages of enlargement of the thyroid may therefore be regarded as the expression of an attempt of the organism to increase its store of iodine. But a lack of iodine will not suffice to account for the causation of goitre, especially in its more severe forms. Even in goitrous regions we find that goitre tends to run in families. Some families are markedly affected, others comparatively little, and others not at all. Numerous family trees have been published, showing the family prevalence of goitre in endemic areas.[1] It is, however, very difficult to decide to what extent family prevalence must be ascribed to hereditary predisposition and to what extent it may depend upon external influences. The fact that in one generation of a family we find some goitrous members and some individuals free from goitre (when all have been exposed to the same environmental influences) suggests that particular hereditary factors play a part. In Fig. 100, I append part of such a family tree published by Bauer.

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[1] Die Erblichkeit des sporadischen Kropfes, "Zeitschrift für induktive Abstammungs- und Vererbungslehre", 1917.

[2] Zur Erblichkeitsfrage des Kropfes, "Archiv für Rassenbiologie", vol. XIV, 1922, no. 1.

The importance of hereditary predisposition as contributing to the causation of goitre even in regions where the malady is endemic, is emphasised by the observations on twins made by Siemens, Pfaundler, and Weitz. We learn from these observations that mon-oval twins are either both free from goitre or both affected with goitre, and that in the latter case they are affected to the same extent. There is no such harmony as regards the incidence or non-incidence of the disease in the case of di-oval twins, or as between mon-oval twins and their brothers and sisters.

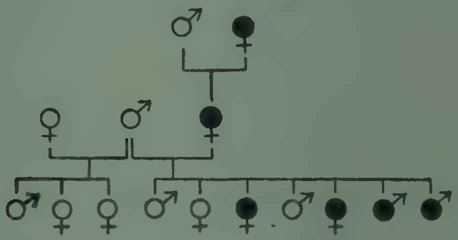

FIG. 100.
Goitre from an endemic area. After K. H. Bauer (extract).

I consider that the clinical material hitherto available suggests that the susceptibility to goitre is not of a uniform kind, and that the causation is polymeric. The heredity does not appear to be sex-linked. Although goitre is much commoner in women than in men, we must suppose that this phenomenon is not dependent upon a sex-linked heredity, but rather upon a greater proneness of the female thyroid to goitrous enlargement. In regions where goitre is markedly endemic, highly developed goitre is only about half as common in men as in women; in regions where goitre is endemic to a lesser degree, well-marked goitre is from four to six times commoner in women than in men; and in regions where goitre only occurs sporadically the affection is ten times commoner in women than in men. Hereditary predisposition to "endemic goitre" and hereditary predisposition to "sporadic goitre" are perhaps of the same kind (Pfaundler, K. H. Bauer); the only difference being that in regions where goitre is endemic a very slight predisposition can become active owing to the intensity of the environmental causes (perhaps a marked lack of iodine), whereas in regions where goitre is not endemic the malady can only make its appearance when the predisposition is very strong and is almost limited to the female sex. I am inclined to suspect that the sodium-chloride content of the food plays a part in the causation of goitre, inasmuch as when considerable quantities of sodium chloride (ordinary table-salt) are ingested, the excretion of iodine salts is more vigorous than when very little table-salt is taken with the food. Animals living in a free state, and therefore upon a diet poor in sodium chloride, do not suffer from goitre, whereas domesticated animals are liable to it. Possibly, therefore, the diffusion of civilisation, accompanied as it has been by a generalisation of the use of the iodine-free rock salt, has contributed to the generalisation of goitre. Of late years, in regions where goitre is endemic, small quantities of iodine have been administered to the population during long periods, and appear to have had good results in the prevention of goitre.

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[1] See M. Pfaundler, Ueber die Entstehungsbedingungen von endemischem Kropf und Kretinismus, "Jahrbuch für Kinderheilkunde", vol. CV, 1924, p. 223; K. H. Bauer, Untersuchungen über die Frage einer erbkonstitutionellen Veranlagung zur Struma nodosa colloides, Bruns, "Beiträge zur klinischen Chirurgie" vol. CXXXV, no. 3, 1926.

There is a fair amount of evidence to show that in the families of immigrants into goitrous regions, goitre is rare and slight in the first generations, and that the disease becomes more severe and more frequent in the course of ensuing generations. Such a sequence of events cannot be explained as due to the working of mendelising genes. We must think, rather, of after-effects (paraphoria), or of plasmatic transmission. Such a view of the matter is confirmed by the circumstance that transmission by the mother seems to be more frequent and more powerful than transmission by the father. The problem of a progressive "goitrous degeneration" is connected with the problem of cretinism.

In certain regions where goitre prevails, we find a considerable number of dwarfed and feeble-minded persons who are distinguished by additional characteristics, such as imperfect development of the reproductive organs, a depression at the root of the nose, etc. These are known as "cretins". *Cretinism* tends markedly to run in families, so that we find, either that several children in series, or all the children of a family, are affected with the disorder. The mothers of cretinoid children always suffer from goitre. I reproduce in Fig. 101 a family tree published by Pfaundler. We have three descriptions of mon-oval twins in which both members of the pair were similarly affected with cretinism.[1]

E. Finkbeiner considers that certain "racial factors", that is to say heredity factors, contribute to the causation of cretinism.[2] But I cannot follow this author in his repudiation of the connexion between goitre and cretinism. Pfaundler, studying 34,500 children, found a correlation coefficient of 0·31 ± 0·00 or a correlation index of 0·50 ± 0·00 for the simultaneous occurrence of goitre and cretinism in a particular individual. The correlation between cretinism in the child and goitre in the mother appears to be even more marked. In cretinism, too, the notable predominance of maternal influence, the occurrence of the malady in a number of successive members of the same family, and the way in which the condition becomes aggravated as the generations pass, speak in favour of plasmatic transmission (paraphoria); but hereditary predisposition in the narrower sense of the term must be a contributory cause.

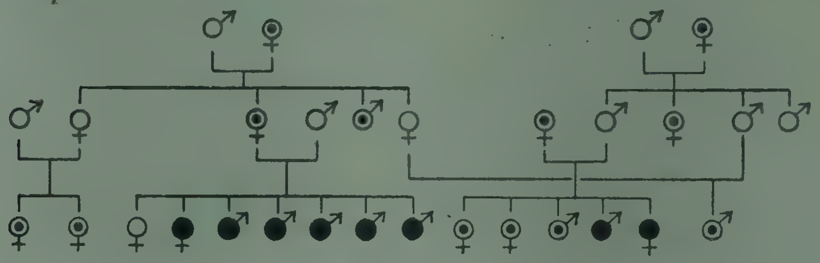

FIG. 101.

Cretinism, after Pfaundler (modified).

The individuals denoted by a circle with a black dot in it suffered only from goitre.

Sporadic cretinism is the outcome of a failure of thyroid function. It is, therefore, a contrasted disturbance to hyperthyroidism. In some of the cases, the thyroid is absent at birth, and in others the gland atrophies in early childhood. Here hereditary predisposition is manifestly the decisive factor.[1] The consequences vary in severity according as to whether the thyroid failure occurs earlier or later in life. Good results can be obtained by the administration of thyroid extract derived from one of the lower mammals. In this way the dwarfing and feeble-mindedness which would otherwise be unavoidable can to some extent be prevented.

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[1] Pfaundler, op. cit.

[2] Die kretinische Entartung, Springer, Berlin, 1923.

Following the terminology introduced by J. Langdon Down, we give the name of "mongolism" to a condition of high-grade feeble-mindedness, in which the outward aspect of the children affected is a caricature of the Mongolian type. I fancy that the morbid Mongoloid has not in most cases been clearly distinguished from the true Mongolian type, which is widely distributed throughout Europe. When idiocy arising from one cause or another occurs in a person of the Mongolian type, the name of "mongolism" is apt to be applied. Still, there seems to be no doubt that there are good biological reasons for distinguishing a Mongoloid type of idiocy. Comparatively seldom has the occurrence of this type been noted in several members of the same family. Davenport records six instances in which only one of a pair of twins was affected.[2] This strongly suggests that special heredity factors must be at work. Siemens quotes a case recorded by Siegert in which, of two mon-oval twins, only one was Mongoloid. This instance does not invalidate the idea of special heredity factors, for in Siegert's case the fact that the twins were of the same sex seemed to him sufficient reason for regarding them as mon-oval. But as regards the origination of mongolism, certain influences proceeding from the mother would seem to be of even greater importance than hereditary predisposition. Mongoloid idiots are usually the offspring of comparatively elderly mothers who have already borne a considerable number of children.[3] The last-born of a very large number of children runs a very high risk of being a Mongoloid idiot. We think, in this connexion, of an exhaustion of the hormonal organs of the mother. Davenport suspects a connexion with inadequate functioning of the maternal thyroid. This would imply a close connexion between mongolism and cretinism—a connexion which most authors refuse to admit. It is, however, a remarkable fact that cretinism occurs chiefly in populations containing a strong Mongoloid admixture (the Alps and the other mountain regions of central Europe, the plain of the Po, the mountains of central and eastern

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[1] H. Zoepffel, Familiäres kongenitales Myxödem, "Zeitschrift für Kinderheilkunde", vol. XXXVI, 1922, no. 4.

[2] C. B. Davenport and G. Allen, Family Studies in Mongoloid Dwarfs, "Journal of Psycho-Asthenics", vol. XXIX, 1925.

[3] W. Weinberg, Methoden und Technik der Statistik, in Gottstein, Schlossmann, and Teleky's Handbuch der sozialen Hygiene, Springer, Berlin, 1925, vol. I, p. 96.

Asia, the mountains of North and South America). V. Lebzelter publishes a map showing the distribution of goitre.[1]

As an appendix to my account of the diatheses manifesting themselves in childhood, I must discuss the *preponderant mortality of male children*, which is likewise mainly determined by a complex of heredity factors. To illustrate the phenomenon in question, I give here the infantile mortality in Germany during the eleven years 1904–1914:

	Of 100 born alive, there died during the first year		Percentage ratio of boy mortality to girl mortality
	Boys	Girls	
1904	21·2	18·0	117·8
1905	22·2	18·8	118·1
1906	20·1	16·9	118·9
1907	19·1	16·0	119·4
1908	19·4	16·2	119·8
1909	18·4	15·4	119·5
1910	17·6	14·7	119·7
1911	20·7	17·7	116·9
1912	16·0	13·4	119·4
1913	16·4	13·7	119·7
1914	17·7	14·9	118·8

We see that during the first year of life the deaths of males exceeded the deaths of females by nearly one fifth. The mortality statistics of all other countries are similar in this respect. The predominant mortality of male infants is to be explained by the existence of recessive sex-linked mobrific heredity factors or diatheses in all populations.[2]

The conditions correspond closely with those met with in certain breeding experiments upon the fruit-fly Drosophila. As was shown in Section One, when a red-eyed male fruit-fly was crossed with a female fruit-fly carrying the white-eye producing factor in a marked form, the theoretical expectation would be, that half of the male offspring would be red-eyed and half of them white-eyed. In carefully conducted experiments, like those of G. Just,[1] such results are approximately obtained. However, the first breeding experiments of this kind, those conducted by T. H. Morgan,[2] resulted in the production of only about three fourths as many white-eyed males as red-eyed males. Here, the experimental conditions having been less favourable, a larger proportion of insects carrying the white-eye factor than of insects carrying the red-eye factor had perished in the larval stage. In P. W. Whiting's experiments, an even larger proportion of the white-eyed males succumbed.[3] Thus, when the environmental conditions are unfavourable, the larvae carrying the white-eye factor, being less hardy, perish in greater numbers than the larvae carrying the red-eye factor; but when the conditions are more favourable, this preponderant mortality of the larvae carrying the white-eye factor does not occur.

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[1] Konstitution und Rasse, in Brugsch and Lewy's Biologie der Person, vol. I, 1926, p. 749.

[2] F. Lenz, Die Uebersterblichkeit der Knaben im Lichte der Erblichkeitslehre, "Archiv für Hygiene", vol. XCIII, 1923, p. 126.

When, in human beings, the infantile mortality due to external noxious influences increases (as happens especially in hot summers, like that of the year 1911), the excess of male infantile mortality over female is less marked. From this we see that the excess of male infantile mortality is mainly determined by internal (hereditary) causes. In conformity with this we find that in hot countries, where infantile mortality is comparatively high—as in Italy and Spain—the preponderance of male infantile mortality over female is comparatively small; whereas in colder lands, where the infantile mortality is comparatively small—in the countries of northern Europe, for instance—the preponderance of male infantile mortality over female is great.

In like manner, the excess of males over females in abortions and miscarriages must be dependent upon recessive sex-linked heredity factors.

We may presume that lethal heredity factors, rendering life impossible even when the environmental conditions are extremely favourable, must account for part of the preponderance of male infantile mortality. Though infrequent, lethal heredity factors could easily account for such a preponderance of male infantile mortality as is actually observed. If we assume that in the human species each chromosome contains on the average one hundred genes, the actually observed preponderance of male infantile mortality would be such as we should have to expect if only one in four thousand genes were recessively lethal.

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[1] Der Nachweis von Mendelzahlen bei Formen mit niedriger Nachkommenzahl, "Archiv für mikroskopische Anatomie", 1920.

[2] Sex-Limited Inheritance in Drosophila, "Science", 1910.

[3] Viability and Coupling in Drosophila, "American Naturalist" 1913.

Of course morbid heredity factors often lead to death in females as well as in males, and in later life as well as in infancy and childhood. Contemplating this from another standpoint we may say that the duration of life is in large measure determined by hereditary predisposition. A. Ploetz,[1] dealing with material comprising 5,585 children, found that their mortality up to the age of five was less in proportion as their parents lived to a greater age. When the mothers or the fathers attained an age of over eighty-five years, the mortality of their children was only from one third to one half as great as the average (11·2 as compared with 32 % or 15·4 as compared with 31·3 %).

Karl Pearson and his pupil Miss Beeton,[2] dealing with material comprising several thousand cases, have found that among adults likewise the expectation of life is greater on the average in proportion to the age attained by the parents of the person concerned. A. G. Bell, dealing with a kinship comprising several thousand persons, obtained similar results.[3] R. Pearl gives a composite picture of what is known regarding the heredity of the duration of life.[4]

It is obvious that the duration of life cannot be monomerically hereditary—that long-life cannot be dominant over short-life. The predisposition which, in conjunction with environmental influences, is decisive as to the duration of life, must, indeed, be markedly polymeric. If in the complex of an individual's heredity factors there were no morbid components, and if he could be successfully sheltered from external noxious influences, he would probably live to an age far beyond a hundred—and we know that, even under present conditions, centenarians are by no means unknown. Most of us, however, have numerous and very diversified morbific heredity factors in our make-up, with the result that, quite apart from the effect of external noxious influences, in one of us one organ and in another of us another will prematurely break down. Let us now, therefore, proceed to discuss such morbific heredity factors, insofar as we know anything about them.

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[1] Lebensdauer der Eltern und Kindersterblichkeit, "Archiv für Rassen- und Gesellschaftsbiologie", 1909.

[2] M. Beeton and K. Pearson, On the Inheritance of the Duration of Life, and on the Intensity of Natural Selection in Man, "Biometrika", vol. I, 1901, p. 50.

[3] The Duration of Life and Conditions associated with Longevity, a Study of the Hyde Genealogy, Washington, 1918.

[4] The Biology of Death, Lippincott, Philadelphia and London, 1922.

Among hereditary disorders and diseases, *haemophilia* has attracted special attention. Literally translated, the word haemophilia signifies "blood-love". Persons affected with this diathesis are known as "bleeders". Whereas in normally constituted individuals, bleeding from a slight wound is soon brought to a stop by coagulation of the blood, in bleeders this fails to occur. Even from a trifling scratch, blood may continue to ooze for many days, thus imperilling life. The extraction of a tooth may be followed by fatal haemorrhage. After a very slight sprain there may be an extensive effusion of blood into the joint cavity, with the result that the affected limb is put out of action for a long time. A considerable proportion of bleeders perish during the first decade of life. After the age of forty, the liability to die from haemophilia is said to decline. We do not as yet know upon what the deficient coagulability of the blood actually depends.

True haemophilia has hitherto been observed only in males. In the women belonging to bleeder families, excessive haemorrhage is no commoner than in other women, not even during menstruation or after childbirth. Bleeders always inherit their malady from their mothers, who themselves remain immune. The taint can be transmitted through the female line for several generations. Women who thus transmit the taint used to be called "conductors". On the other hand, the direct transmission from father to son has never been observed. Transmission from grandfathers to grandsons by way of females has at length been clearly established by H. Schloessmann through a comprehensive study of bleeder families in Wurtemberg.[1] The difficulty in proving this has been due to the fact that bleeders so seldom grow up, marry, and procreate children.

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[1] Die Hämophilie in Württemberg, genealogische, erbbiologische und klinische Untersuchungen an 24 Bluterfamilien, "Archiv für Rassenbiologie", vol. XVI, 1924, nos. 1 to 4.

We see, then, that haemophilia is recessively sex-linked. An extract from a family tree illustrating this is reproduced in Fig. 102.

K. H. Bauer[1] advocates the view that the bleeder taint is recessively lethal in females. But there is no reason to make any such assumption, seeing that the absence of female bleeders among the offspring of the few marriages between bleeders and conductors hitherto recorded is easily explicable in other ways. Bauer has also identified with the normal gene allelomorphic with the heredity factor producing haemophelia what he terms "thrombokinasis"—a hypothetical enzyme which he supposes to be the cause of the coagulation of the blood. Even if "thrombokinasis" were anything more than a word, its identification with a gene would be untenable.

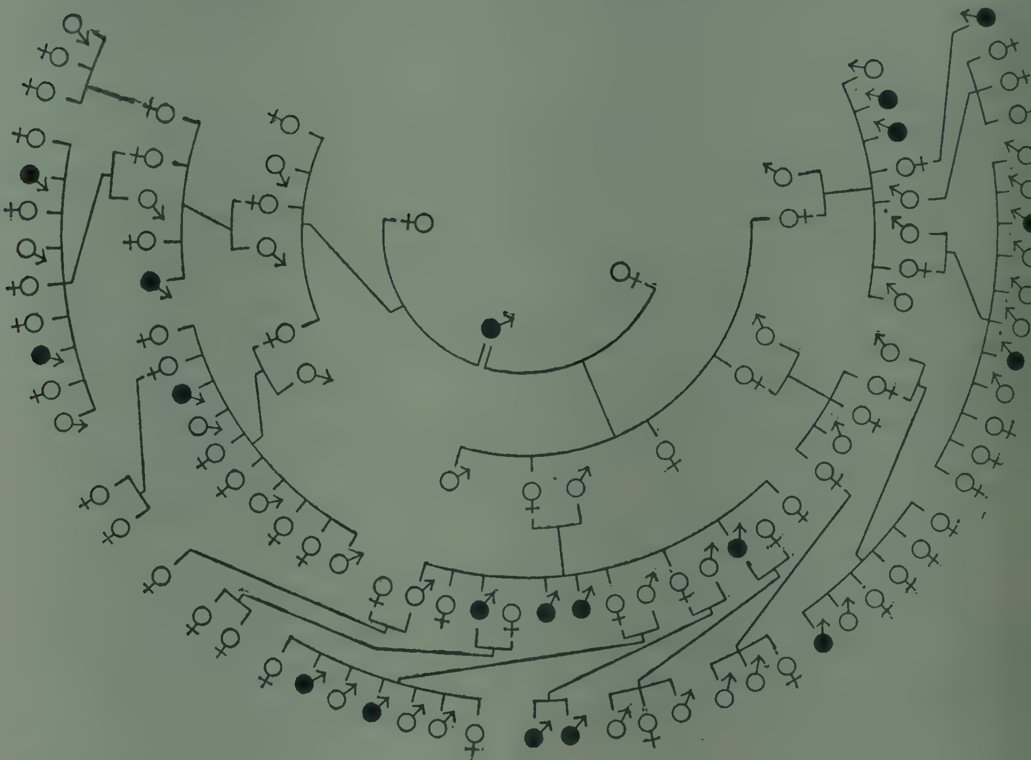

FIG. 102.
Haemophilia, after Schloessmann (extract),

Schloessmann's investigations have shown that in "conductors" (women who carry the bleeder taint heterozygotically) coagulation of the blood is somewhat delayed, and that they occasionally suffer rather unduly from haemorrhage, though never so severely as male bleeders.

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[1] Zur Vererbungs- und Konstitutionspathologie der Haemophilie, "Deutsche Zeitschrift für Chirurgie", vol. CLXXVI, 1922, p. 109.

Haemophilia is not met with everywhere. It is especially prevalen in certain regions, as, for instance, Wurtemberg and Switzerland In some of the bleeder families of the United States, a hereditary connexion can be traced with bleeder families in Europe.

It has been contended that bleeder families are exceptionally prolific, and that among the children of such families there is an exceptionally large proportion of boys. It is also said that in bleeder families among the male offspring there are many more bleeders than non-bleeders. These contentions are erroneous, being based upon the application of unsound methods of calculation (see below, page 506).

For a long time the bleeder families of Mampel, described by Lossen, remained classical as an illustration of the hereditary transmission of disease. The novelist Ernst Zahn has made the history of the bleeders of Tenna (in Grisons, Switzerland) the theme of his novel *Frauen von Tannò*; but he gives an incorrect account of the heredity of haemophilia. Hanhart has recently amplified the genealogical trees of the Tenna bleeders, so that this is now the most extensive of all the bleeder family trees. In the present generation of this family there have been three instances in which bleeders were born as the grandchildren of bleeders. All the older family trees of haemophilia have been republished in the "Treasury of Human Inheritance".[1]

We must distinguish from true haemophilia a less frequent hereditary tendency to bleeding (the haemorrhagic diathesis) which, in contradistinction to true haemophilia, manifests itself more commonly in females than in males, and is more severe in women than in men. E. A. Willebrand[2] has reported upon it under the name of "hereditary pseudo-haemophilia". I reproduce in Fig. 103 the family tree he publishes illustrating this malady. Willebrand opines that we have to do with a dominant sex-linked heredity. We see in the left upper part of the tree several cases in which bleeders' children appear to have been the offspring of healthy parents, and this is inconsistent with the assumption of genuine dominance, but the probable explanation is that we are inadequately informed concerning these parents. Among the bleeders in this kinship, the manifestation of the diathesis usually took the form of nose-bleed, haemorrhage from the gums, haemorrhagic extravasations in the skin; and in the female members (in addition to the foregoing) of post-partum haemorrhages and of intestinal haemorrhages. It is likely enough that in earlier generations a proneness to nose-bleed or to haemorrhage from the gums may not have been regarded as the manifestation of a morbid diathesis. The observations recorded in the right-hand lower part of the tree are less compatible with the idea of a dominant sex-linked heredity. If the father had really been the carrier of the taint, we should have expected that all the brothers would have been affected. Actually, however, two of the eleven daughters appear to have been healthy (the youngest was only eighteen months old at the time of the investigation). Of the father, nothing more is recorded than that he frequently suffered from severe nose-bleed. If he were really the carrier of the taint, we must dismiss the assumption of a sex-linked heredity, and must suppose an incompletely dominant heredity of the ordinary kind. The greater danger to the female sex must doubtless be connected with the female reproductive processes, seeing that even a normal childbirth involves the loss of a good many ounces of blood. In the family described by Willebrand, five women perished from haemorrhage. Unfortunately, the coagulation time of the blood does not seem to have been recorded in the members of this family, and this is a point for further investigation, especially as concerns the father of the last family. It might thus be possible to find the solution of this interesting problem.

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[1] W. Bulloch and P. Fildes, Haemophilia, in "Treasury of Human Inheritance", parts V and VI, Cambridge University Press, London, 1911.

[2] Hereditär Pseudohemofili, "Finska Läkaresällskapets Handlingar", vol. LXVIII, 1926, p. 87.

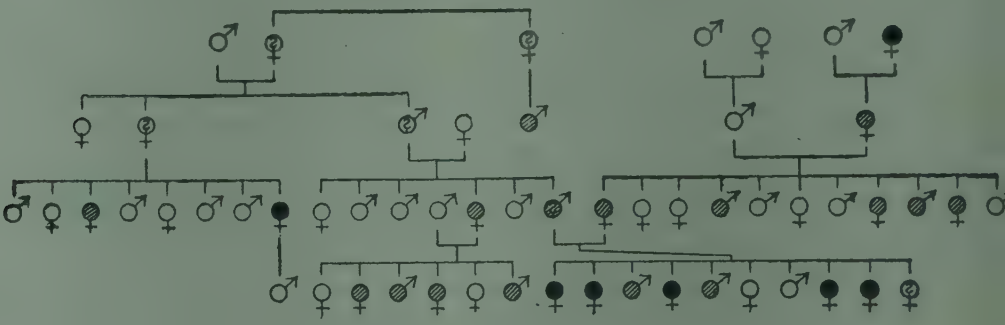

Fig. 103.

Pseudo-haemophilia, after Willebrand.

Black circles denote a severe tendency to bleeding; obliquely hatched circles denote a slight tendency.

Some of the earlier family trees recorded in the "treasury" would seem to relate to Willebrand's pseudo-haemophilia, and the same remark applies to the trees recently published by Glanzmann[1] and Meumann.[1] We may also assign to this hereditary haemorrhagic diathesis a considerable proportion of the cases classed under the caption "morbus maculosus Werlhofii" (purpura haemorrhagica).

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[1] Hereditäre hämorrhagische Thrombasthenie, "Jahrbuch für Kinderheilkunde", vol. LXXXIII, 1918, p. 271.

A hereditary diathesis which is of great practical importance is the constitutional *hypertension* or *hypertonia* which manifests itself as an abnormally high blood-pressure. In a good many of those who are affected with a morbidly high blood-pressure, the renal arteries eventually become hardened, the normal tissue in the walls of the bloodvessels being replaced by connective tissues. As a result of this nephrosclerosis, there ensues a further increase in blood-pressure. In consequence of these changes, considerable areas of the proper renal tissue perish, and there is produced the contracted kidney, which is always of extremely grave significance. When we find a family tendency to the formation of contracted kidney—in the absence of other disorders and of toxic causes (syphilis, gout, lead-poisoning)—we are led to think of hereditary hypertonia. Heredity, likewise, must account for the family prevalence of apoplexy and heart attacks at a comparatively early age (the sixth decade of life), if no accidental noxious influences such as syphilis, alcoholism, or nicotinism, explain the condition. Apoplectic strokes result from rupture of sclerosed cerebral arteries; heart attacks (angina pectoris, etc.) usually depend upon sclerosis of the coronary arteries, which provide the heart with blood. Many hypertonics die from simple heart failure (heart insufficiency).

The hereditary determination of hypertension has been mainly elucidated by Professor W. Weitz of Tübingen.[2] Weitz sums up as follows the outcome of his investigations concerning the causes of hypertension. "It seems to me beyond question that a dominant heredity accounts for the majority of the cases. For most of the other cases a dominant heredity is probable but not certain; and there is a small residue of instances in whose production heredity has not been shown to play a part." Certain external noxious influences, such as alcoholism and nicotinism, have hitherto been regarded as important causes of high blood-pressure, but in Weitz's opinion these are of little moment. Women suffer from hypertension about as frequently as men. Mon-oval twins resemble one another strikingly in this matter of blood-pressure;[1] especially remarkable was the case of mon-oval twin sisters, one of whom had throughout life been engaged in arduous physical toil in factories and on the land, whereas the other had lived in easy circumstances in towns. Nevertheless, at the age of sixty-four both of them were affected to the same degree by a morbidly high blood-pressure.

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[1] Echte Hämophilie beim Weibe, "Zentralblatt für Gynäkologie", 1922, p. 590.

[2] Zur Aetiologie der genuinen oder vaskulären Hypertension, "Zeitschrift für klinische Medizin", vol. XCVI, 1923, nos. 1 to 3.

Weitz has been good enough to communicate to me the family tree which I reproduce in Fig. 104.

At the time of the investigation the blood pressure in the first six members of the second generation, as measured in millimetres of mercury by Riva-Rocci's manometer, was as follows: 1, aged 57, b.p. 200 mm.; 2, aged 54, b.p. 180 mm.; 3, aged 52, b.p., 130 mm.; 4, aged 50, b.p. 185 mm.; 5, aged 44, b.p. 120 mm.; 6, aged 42, b.p. 160 mm. (In healthy persons the blood-pressure does not exceed 140 mm.) In the seventh member of the family (second generation) there was no morbid increase in blood-pressure. The father had died of heart trouble at the age of seventy-one, and the mother of "dropsy" (presumably due to heart disease or kidney disease) at the age of sixty-one.

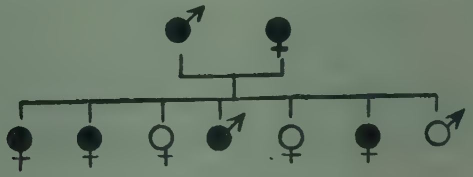

Fig. 104.
Morbidly high blood-pressure (after Weitz).

Weitz, as a result of the study of the material he collected, was unable to confirm the traditional view that morbidly high blood-pressure occurs chiefly in persons of a "stocky" type—persons with an "apoplectic habit of body".

Abnormally high blood-pressure is one of the main causes of *arteriosclerosis*. The latter disease manifests itself as localised patches of induration in the arteries, these patches ultimately becoming calcified. In a sense we may say that the arteries become prematurely worn out. Although many persons who are well advanced in years are entirely free from arteriosclerosis, there are extensive variations in the age at which the trouble begins and in the parts of the arterial system which are chiefly affected. External noxious influences (occupational and toxic) unquestionably play a part in its causation. At the same time, hereditary predisposition is manifestly of extreme importance here. That is why arteriosclerosis has so marked a tendency to run in families. In certain families, the liability to arteriosclerosis is of a general character; in others, we note a special tendency to arteriosclerosis of the brain, with consequent attacks of apoplexy; in others, again, the predominant tendency is to sclerosis of the coronary arteries, causing heart trouble; and in yet others, it is the renal blood vessels, above all, which become affected. These distinctive trends are the outcome of a general inclination towards hypertonia, associated in one family with an inclination to the localisation of the arteriosclerosis in one organ, and in another family in another organ.

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[1] W. Weitz, Studien an eineiigen Zwillingen, "Zeitschrift für klinische Medizin", vol. CI, 1924, nos. 1 and 2.

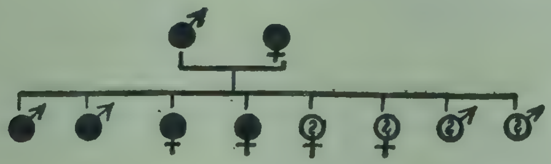

FIG. 105.
Apoplexy, after Weitz.

Fig. 105 shows a family in which the first four members of one generation of eight brothers and sisters died of apoplexy between the ages of sixty and seventy years. Both of the parents had also died of apoplexy. The four younger members of the second generation were still living at the time when the observations were made, but it is to be presumed that one or more of them will also die of apoplexy.

Although some observers have declared arteriosclerosis to be commoner in Jews that in Gentiles, according to the researches of H. von Ullmann this does not appear to be the case.[1]

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[1] Zur Frage der Vitalität und Morbidität der jüdischen Bevölkerung, "Archiv für Rassenbiologie", vol. XVIII, 1926, no. 1, p. 1.

We give the name of metabolic disorders to a group of diatheses in which disorders of metabolism (tissue change) occupy the foreground of the picture. Speaking strictly, indeed, all diatheses are associated with anomalies of the process of tissue change. On the other hand, every metabolic disorder is a constitutional disorder, and every diathesis is a constitutional anomaly—the various concepts shade into one another. But among metabolic disorders in the narrower sense of the term we include diabetes, obesity, and gout.

Diabetes (in full, diabetes mellitus) is characterised by the excretion of grape sugar in the urine. The diabetic patient has partly lost the capacity of making an effective use of sugar and other carbohydrates, which normally constitute a major part of the food and provide a major part of the calorific energy required by the animal body; and, in especial, the diabetic has lost the power of storing up carbohydrates in his body. Besides the loss of energy which results from incomplete utilisation of his food, the diabetic suffers from and has his life endangered by various other consequences of the disorder of metabolism. For instance, he is liable to auto-intoxication from undue formation of acids within his system (acidosis).

Diabetes usually makes its appearance in middle life, and often not until the patient is well advanced in years. It depends upon a functional failure of certain cell groups in the pancreas (the so-called islands of Langerhans); that is to say, upon a disturbance of internal secretion. There can be no doubt that in many cases, the failure of these cells is the outcome of a hereditarily determined weakness. In other cases, however, external noxious influences are the predominant cause, especially syphilis and chronic intoxication of various kinds. Diabetes is more than twice as common in men as in women, this being due, not to sex-linked heredity, but to the predominant exposure of males to the before-mentioned external noxious influences. In conformity with this we find that down to the close of the third decade of life the liability of females to diabetes is as great as that of males. For in youth diabetes is hardly ever due to external causes. From the fourth decade of life onwards, external influences come to play an increasing part in the causation of the disease, this accounting for the preponderant incidence among males in the later decades of life. Diabetes being more often hereditarily determined in women than in men, we can understand why females more frequently inherit the predisposition than do males. Probably this difference is nowise dependent upon a sex-linked heredity. Another way of looking upon diabetes is to regard it as a premature senescence of the islands of Langerhans, comparable to the premature greying of the hair in many families. In different persons the different organs age with varying rapidity, and the process of ageing in any organ is accelerated by making excessive demands upon it. In certain families diabetes seems to have a dominant heredity, as we see in a family tree published by Noorden[1] (Fig. 106). F. A. Long[2] publishes three carefully compiled family histories. J. A. Buchanan[3] was able to trace the occurrence of diabetes in seventeen families for three generations.

S. Hansen[4] is strongly of opinion that in cases of diabetes hereditary influences can be shown to be at work far more often than has hitherto been supposed. In Fig. 107 I reproduce one of his family trees. Hansen believes that the causation is polymeric; but in my view the reasons he adduces are insufficient to justify the exclusion of a far more probable monomeric heredity. At any rate, a simple dominant heredity does not appear to be the rule. If we are very seldom able to trace the inheritance of diabetes through several generations, this is presumably because in earlier generations there was often a failure to recognise diabetes, and also because many carriers of the taint died from some other cause before the outbreak of the disease. Considering the recorded experience as a whole,

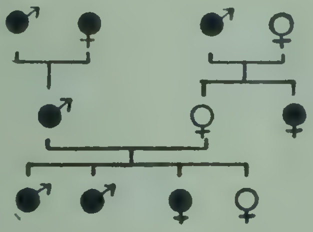

FIG. 106.

Diabetes, after Noorden.

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[1] Die Zuckerkrankheit, Berlin, 1910.

[2] A Contribution to the Study of the Familial Aspects of Diabetes Mellitus, "Western Medical Review", vol. XXX, no. 19.

[3] A Consideration of the Various Laws of Heredity and their Application to Conditions in Man, "American Journal of the Medical Sciences", vol. CLXV, 1923, no. 5.

[4] Ueber die Vererbung des Diabetes Mellitus, "Acta Medica Scandinavica", vol. LXII, 1925, nos. 1 and 2.

it seems to me that in most cases diabetes is caused by recessive heredity factors. There are varying reports as to the frequency of consanguinity in the parents of diabetics.[1] Inasmuch, however, as we have to do here with a very frequent taint, even if the heredity be recessive we should not expect to find a high percentage of consanguineous marriages among the parents of diabetics (see below, page 524). To some extent, likewise, the comparative frequency of diabetes in Jews speaks in favour of the notion that the heredity is recessive.

In the case of diabetes as in the case of certain other diseases, several authors have described what they term "anticipation", this meaning that the disease is supposed to appear earlier and earlier in successive generations. But inasmuch as in one and the same family diabetes may manifest itself at various ages, the result will in general be that among the diabetics of an earlier generation, those only in whom the disease was of comparatively late onset will have married and have had children. Diabetic women are usually sterile; and even diabetic men, owing to the sexual weakness associated with the disease, seldom procreate children. In this way the parents are selected from among those in whom the disease appears comparatively late in life. On the other hand, in the most recent generations, we shall find as diabetics only the individuals in whom the trouble has come on early in life, whereas their brothers and sisters in whom the manifestation of the taint is deferred will be accounted healthy at the time of the investigation. Another point to be borne in mind is that in the offspring of diabetics the presence of the illness is often detected at an earlier age than it was

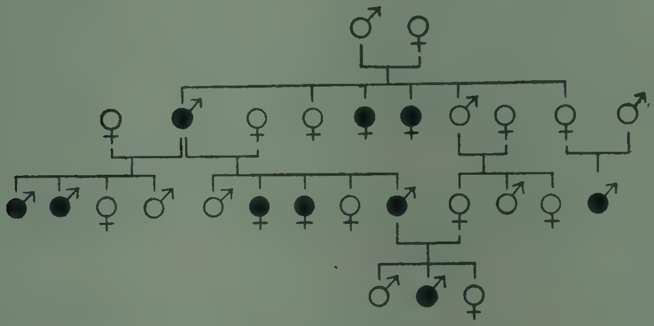

FIG. 107.
Diabetes, after Hansen.

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[1] N. B. Foster, Consanguineal Diabetes Mellitus, "Bulletins of the Johns Hopkins Hospital", vol. XXXIV, 1912, no. 23.

in the parent, for the obvious reason that people are on the look-out for it. This contributes to produce a semblance of "anticipation".

Hereditarily determined diabetes is in Central Europe much commoner among Jews than among the Gentile population. According to Theilhaber, in Berlin during the year 1910 eighty adult Jews died of diabetes. From the Statistical Year-Book of the town of Berlin we learn that in that city during the year 1910 the total number of deaths from all causes among male Jews of more than fifteen years of age was 546. If we assume the sex incidence of the disease to have been the usual one of three males to two females, 48 of the 546 males who died must have died of diabetes, this being nearly 9 %. In the general population, on the other hand, among 9862 deaths of adult males, only 232 deaths were due to diabetes, this being a percentage of 2·4. Deducting the deaths of Jews, we get a percentage of deaths in the Gentile population of no more than two. Were it possible to separate the baptised Jews from the other Christians, the percentage of deaths among non-Jews would of course be smaller still. On the whole it would seem that among Jews diabetes is a cause of death about six times as often as among Gentiles. The previously quoted work by H. Ullmann contains all the relevant statistical material. Noorden considers that the preponderant liability of Jews to diabetes is due to the greater frequency of the marriage of near kin among Jews. Furthermore, however, there is good reason to believe that the heredity factors of diabetes are actually more prevalent in the Jewish than in the Gentile population.

We must be careful to distinguish from true diabetes what is known as "*renal glycosuria*", wherein sugar is likewise excreted in the urine. Renal glycosuria, however, is comparatively harmless, being apparently due to a non-progressive anomaly of the kidneys. This disorder is likewise hereditary, and it would seem that the heredity is dominant.[1] Even in true diabetes, according to B. Naunym[2] and H. Seckel,[3] the cases in which heredity can be traced through two or more generations are in general comparatively mild. Here is another confirmation of the rule that dominant disorders are apt to run a milder course than do kindred troubles in which the heredity is recessive.

What is known as *diabetes insipidus* has only this much in common with diabetes mellitus, that in both disorders excessively large quantities of urine are excreted. In diabetes insipidus, there is no sugar in the urine. Owing to the voluminous excretion of water, great thirst ensues, and is a torment to the patient unless relieved by drinking vast quantities of fluid. The disease is also known as hereditary polyuria. A number of genealogical trees have been drawn up, and they show the heredity to be dominant.

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[1] T. Brugsch and K. Dresel, Renale hereditäre Glycosurie, "Medizinische Klinik", 1919, no. 39.

[2] Der Diabetes Mellitus, second edition, Vienna, 1906.

[3] Beobachtungen über heredofamiliäre und konstitutionelle Häufung von Stoffwechselleiden beim Diabetes mellitus, "Zeitschr. f. klin. Medizin", vol. CII, 1925, p. 195.

A large genealogical tree published by A. Weil[2] has become classical because of the clear way in which it manifests a dominant heredity. Since it has several times been inaccurately reproduced, G. Just[3] has recently printed it once more, and correctly. Among brothers and sisters 33 were diseased and 30 were healthy, which closely corresponds to the theoretical ratio of 1 : 1. Only from considerations of space do I refrain from publishing this tree in the present work.

Cystinuria or the cystin diathesis is the outcome of a hereditary disturbance of proteid metabolism. The normal break-up of the sulphur-containing constituent of protein is interfered with, and this constituent makes its appearance in the urine in the form of cystin, a sulphur-containing amino-acid. Cystin calculi may form in the bladder. That is the most serious outcome of this rare condition, but in other respects it is by no means harmless. The taint appears to be dominant. *Alcaptonuria* is another result of disturbance of proteid metabolism. In this instance there is a hindrance to the break-up of the benzol nucleus of proteins, with the result that homogentisin acid—a normal intermediate product of the break-up of proteins, containing the benzol nucleus—appears in the urine. Urine containing this substance becomes dark-brown or black after exposure to the air. Staining and deposits also occur in various organs of the body. According to E. Toenniessen,[4] the taint is simply recessive.

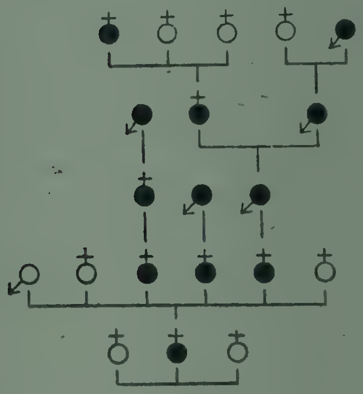

FIG. 108.

Diabetes insipidus, after Gänsslen and Fritz[1] (extract).

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[1] Ueber diabetes insipidus, "Klinische Wochenschrift", third year of issue, 1924, no. 1.

[2] Ueber die hereditäre Form des Diabetes insipidus, "Deutsches Archiv für klinische Medizin", vol. XLIII, 1908, p. 181.

[3] Ein Wort zu Weils Diabetes insipidus Stammbaum, "Archiv für Rassenbiologie", vol. XVI, 1925, no. 3.

[4] Ueber die Vererbung der Alkaptonurie, "Zeitschr. f. indukt. Abst.- u. Vererbungslehre", 1922.

Obesity manifests itself as the deposit of abnormal quantities of fatty tissue beneath the skin of the belly, the loins, the thighs, etc. In the graver forms of obesity, the body weight may be as much as 20 stone or more; but even in the slighter forms, obesity cannot be regarded as harmless, for Florschütz has shown that obese persons have a reduced expectation of life. Diet, of course, plays a considerable part in the production of obesity, which cannot develop in cases of chronic underfeeding. On the other hand, those who have a constitutional tendency to be thin cannot be made obese by overfeeding. Neither gluttony nor sloth can unaided lead to obesity. All the varieties of obesity are associated with a disturbance of internal secretion. In some of the cases of this disorder there has been a decline in the functional activity of the thyroid, which is the regulator of the speed of metabolism and has been compared to a bellows whose purpose it is to fan the fires of the body. Many cases of obesity are obviously dependent upon a defective development of the reproductive glands. A special variety of the disorder is due to defective functioning of the pituitary. For the rest, it would seem that when obesity is marked there is more or less defect of function in all the glands of internal secretion.

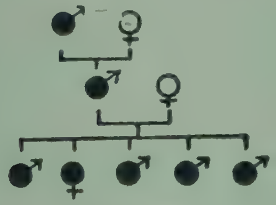

FIG. 109.
Cystinuria, after Abderhalden.

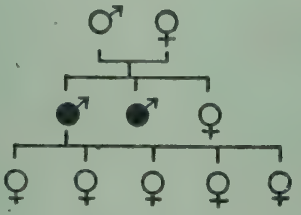

FIG. 110.
Alcaptonuria, after Ebstein.

At the Tübingen polyclinic, Weitz and Liebendörfer[1] have studied the heredity of obesity. In twenty-five cases of obesity studied during the hunger years after the war, in every instance there were other cases of obesity in the family. As a rule the heredity of constitutional obesity appears to be dominant, but in view of the multiformity of the conditions of internal secretion we may presume that there must be heredity factors of obesity in which the heredity is of another type.

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[1] Ueber Erblichkeitsverhältnisse bei Feltsucht, "Archiv für Rassenbiologie", vol. XV, 1923, no. 1.

Of course constitutional leanness is likewise hereditarily determined. In persons having the same body weight and living the same kind of life the amount of food needed may be very different, and there can be no doubt that such differences are hereditary in the main. Many persons inclined to obesity can get along with astonishingly small quantities of food. Lecturing in Munich, Gruber, basing his remarks for the most part upon his own experiments on metabolism, has declared that the intensity of metabolism varies greatly from person to person, being in some only half as great as it is in others.

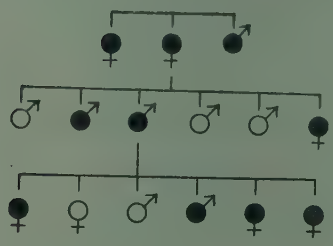

FIG. 111.

Obesity, after Weitz and Liebendörfer.

Before Gruber's researches were made, great uncertainty prevailed as to whether the turnover of nutrition ought to be considered in relation to the body weight, the body surface, or some other standard of measurement. Gruber has shown, however, that the best standard of reference is the stature. In healthy persons of very varying age, stature, and constitution, there is considerable uniformity in the ratio between the tissue turnover and the unit of body length. Whereas, therefore, it has been usual to establish a ratio between the unit of tissue change (the calorie) and the unit of body weight or of body surface, it is better to establish a ratio between the calorie and the unit of body length. When this ratio diverges considerably from the average, we can confidently infer that the intensity of metabolism is abnormal.

When an individual is lean in proportion to his stature or lacks girth in proportion to his stature, people are too ready to infer that he is "under-nourished". This mistake was especially common in investigations upon schoolchildren during and shortly after the war. Although the atrophic condition of German children during this period was doubtless in great measure due to a lack of proper nutriment, there can be no doubt that it was also in great measure due to morbid heredity.

Constitutional obesity and constitutional leanness are plainly related to racial distinctions. Obesity is unquestionably commoner in Chinese, Turks, Magyars, and Jews; leanness is commoner in Anglo-Saxons, Scandinavians, Spaniards, pure-blooded Semites, and the German aristocracy.

It is often asserted that the three great metabolic disorders, diabetes, gout, and obesity, tend to occur in the same families. It has even been supposed that all three of them depend upon the same heredity factor, and are so to say interchangeable. People who hold this view speak of "heterogeneous" or "polymorphous" inheritance. But what we actually find is that in families where the occurrence of metabolic disorders in a severe form can be traced through several generations, the heredity is essentially "homogeneous". On the other hand, we may well suppose that the slighter morbific heredity factors when variously combined may lead to various disorders of metabolism. Thus the trend towards obesity may easily result in an overloading of the body with carbohydrates. In this way, a minor weakness in carbohydrate metabolism which would otherwise have no morbid consequences can manifest itself as diabetes. Various relationships of this kind are conceivable.

Gout (arthritis urica) is characterised by attacks of extremely painful inflammation of the joints, wherein there may be deposits of uric acid in the articular cartilages. These deposits of uric acid may grow into large nodules, greatly impairing mobility. Since uric acid is derived from the nucleins in animal cells, the gouty diathesis manifests itself more frequently (attacks of arthritis and articular changes) in proportion as a larger quantity of nuclein-containing animal food is taken. Lead poisoning and the abuse of alcohol are also exciting causes of gouty attacks. Gout usually begins to manifest itself in the fourth decade of life, and it is much commoner in men than in women.

The inheritance of gout is as a rule simply dominant[1] In many families, the gouty diathesis can be traced through several generations; in other families, the course of the heredity appears to be interrupted, this perhaps being because external influences (and especially the nature of the food) are of great importance as exciting causes of attacks of gout. Fig. 112 and Fig. 119 illustrate the heredity of gout. The disease is especially common among Nordics (the coastal regions of the North Sea and the Baltic).

The inclination to the formation of *renal calculi* is closely connected with the uric-acid diathesis. Uric-acid stones in the kidney and in the bladder respectively, are often found in members of the same family, and in such families we are apt to find that there are also persons suffering from gout.[1]

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[1] W. Ebstein, Die Natur und Behandlung der Gicht, Wiesbaden, 1906.

There is also said to be a family proclivity to the formation of oxalate calculi in the kidney. The same remark applies to cystin calculi (see above).

In earlier medical literature, especially in France, the notion of "arthritism" or the "arthritic diathesis" plays a great part. This was supposed to be the general foundation of diabetes, gout, obesity, arteriosclerosis, contracted kidney, asthma, psoriasis, muscular rheumatism, and many other troubles. Although heredity is doubtless a part cause of all these conditions which have been lumped together under the head of arthritism, there are no adequate grounds for regarding an "arthritic diathesis" as a biological unity. I may refer back to what was said on p. 331 concerning "combined diathesis".

Gout may be included in the group of the *idiosyncrasies* or *allergies*. These are all characterised by a morbid susceptibility to substances which are harmless to normal individuals. Gout is an idiosyncrasy towards cell nuclei of animal origin. There are many such morbid idiosyncrasies. B. N. Belaieff[2] has described a hereditary idiosyncrasy towards egg albumin. I am indebted to Hanhart of Zurich for the sight of a number of interesting family trees compiled by him showing idiosyncrasies as the outcome of which allergic reactions could be induced by various substances in different members of the same family. The heredity appears to be dominant.

What is known as *hay fever* depends upon an allergy towards the pollen of certain grasses. It is an extremely troublesome affection, manifesting itself above all in the nasal mucous membrane and the conjunctiva. A genealogical tree for which I am indebted to Geheimrat Study of Bonn shows how a taint which on the whole appears to be dominant manifests itself in the earlier members of a family as gout (that is to say, idiosyncrasy towards animal cell nuclei), and in the younger members of the family as hay fever (that is to say, idiosyncrasy towards pollen grains). It seems likely that the younger persons who now suffer only from hay fever will in later life, some or all of them, suffer from gout.

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[1] P. K. Pel, Die Erblichkeit der chronischen Nephritis, "Zeitschrift für klinische Medizin", vol. XXXVIII, 1899, p. 127.

[2] Zur Idiosynkrasie gegen Hühnerei, "Berliner klinische Wochenschrift", 1921, no. 47.

Nettlerash (urticaria) is an allergic reaction of the skin. After the taking of certain kinds of food (strawberries, crab, etc.) weals appear on the surface of the body, and recur in crops for several days. They are attended with intense itching. There seems to be a certain kinship between nettlerash and th epreviously described erythma exsudativum multiforme (see above, p. 273). Akin, likewise, is what is known as *Quincke's oedema* (angioneurotic oedema, or acute circumscribed oedema), which is a much graver disorder than the comparatively harmless nettlerash. In persons liable to this affection there are sudden attacks of circumscribed recurrent swelling of areas of skin. If this oedema is limited to the surface of the body, it is not so serious. But sometimes it affects the mucous membranes, being very troublesome in the nose, and exceedingly dangerous in the larynx, where it may cause death by suffocation. From

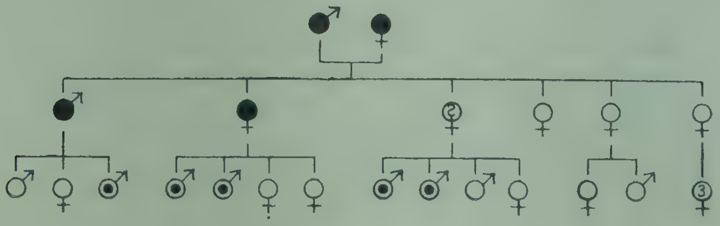

FIG. 112.

Gout and hay fever, after Study.

Black circles signify gout.
Dots within circles signify hay fever.

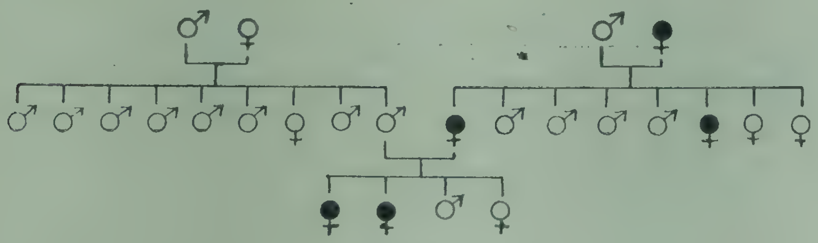

FIG. 113.

Inclination to nettlerash, after Fetscher.

J. R. Crowder and T. R. Crowder[1] I reproduce a genealogical tree showing the heredity of this affection (Fig. 114). J. Philipps and W. Barrows have reported on the heredity of angioneurotic oedema.[2] The earlier genealogical trees have been collected by W. Bulloch.[3] The same individuals, or a number of individuals in the same family, may suffer from hay fever, asthma, nettlerash, and eczema. There also seem to be relations between these disorders, on the one hand, and gout, gall-stones, and

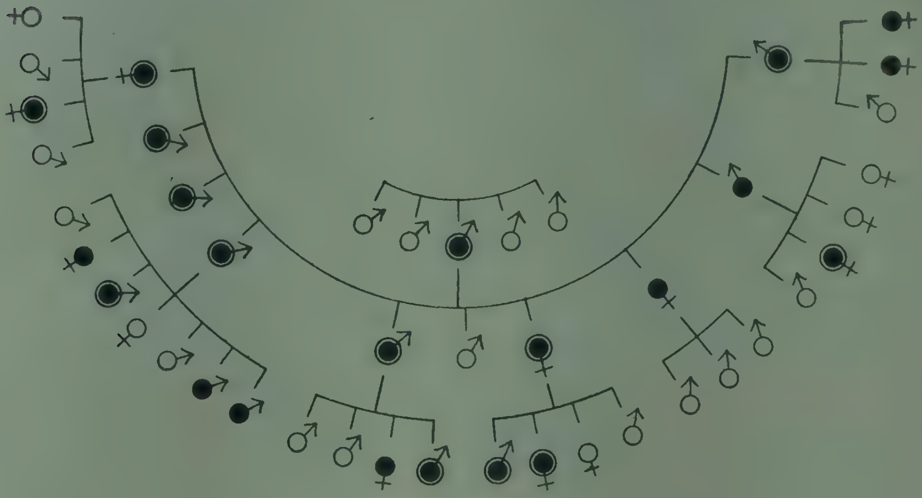

FIG. 114.

Attacks of circumscribed swelling of skin and mucous membranes (Quincke's oedema), after Crowder (extract). Black circles surrounded by a ring denote deaths from laryngeal oedema.

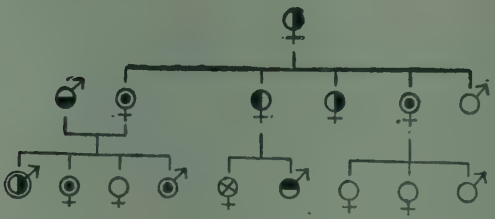

FIG. 115.

Family tendency to various allergies. After Gänsslen (extract).

◒ = gout.
◑ = migraine.
◐ = gall-stones.
◓ = hay fever.
⊗ = asthma.
◉ = nettlerash.
○ = Quincke's oedema.

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[1] Five Generations of Angioneurotic Oedema, "Archives of Internat. Medicine", vol. XX, 1917.

[2] "Genetics", vol. VII, 1922.

[3] Angioneurotic Oedema, "Treasury, etc.", part III, section IXa, Cambridge University Press, London, 1909.

migraine, on the other. A family tree showing a number of different allergies is reproduced, after Gänsslen, in Fig. 115.[1]

Hydrops articulorum intermittens, a periodic swelling of one joint or another (usually one of the knee joints), is probably due to hereditary predisposition. About this affection, however, very little is known.

The hereditary course of true *migraine* can usually be followed uninterruptedly for several generations. This trouble is mainly characterised by the sudden onset of attacks of violent one-sided headache, lasting from several hours to a day. Perhaps the immediate cause of the affection is an attack of circumscribed swelling in the cerebral membranes. For the family tree reproduced in Fig. 116, I am indebted to Dr. Unger, the medical director of a sanatorium at Weilmünster in the Taunus. In this case, as usual in migraine, the heredity is dominant.

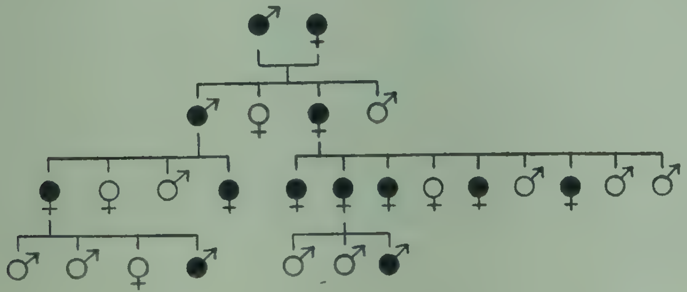

FIG. 116.
Migraine, after Unger.

Inasmuch as migraine occurs about twice as often in women as in men, we must take into account the likelihood that there is a dominant sex-linked inheritance. Unger's family tree is in conformity with this idea except for one point, the exception being that the affected males of the first generation ought to have had affected daughters only. Apart from this, the sexual life seems to have an influence upon the occurrence of migraine. There are women who suffer from migraine only when they are menstruating, and never have an attack during pregnancy. A family tree published by W. Weitz[2] in which migraine is shown to have occurred in twelve individuals belonging to four successive generations, harmonises perfectly with the notion of a dominant sex-linked heredity.

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[1] "Medizinische Klinik", 1921, no. 41.

[2] Die Bedeutung der Erblichkeit für die Aetiologie, "Ergebnisse der gesamten Medizin", vol. V, 1925, p. 468.

Headache of a migrainoid character occurs also in epileptics and in the relatives of epileptics. In these cases, however, we are probably concerned with a hereditary taint differing from that which causes ordinary migraine, with one which is a common foundation for epilepsy and migraine.

To the group of allergic reactions we can also assign *bronchial asthma*, which manifests itself in the form of more or less frequent attacks of severe difficulty in breathing, occurring above all at night. In these attacks, the mucous membrane of the air passages is swollen, and secretes a tenacious mucus. Many investigators regard it as the manifestation of a constitutional hyperexcitability of the vegetative nervous system (the vagal system). Other manifestations of this "vagotonia" are said to be an excessive secretion of acid in the stomach, spasmodic constipation, globus hystericus (the sense of having a ball in the throat), a proneness to perspire, slowness of the pulse, etc. In early childhood, those who are destined subsequently to become asthmatics usually manifest the signs of the inflammatory diathesis in the skin and mucous membranes. In many cases, too, asthmatics have or have had neurotic (it would be better to say, psychopathic) symptoms, but it would be a mistake to regard asthma as nothing more than a form of hysteria. The paroxysm may be induced by overexertion, chills, excitement, etc. In some asthmatics, the attacks are caused by the inhalation of particular substances—the smell of horses, for instance. In this respect, asthma is akin to

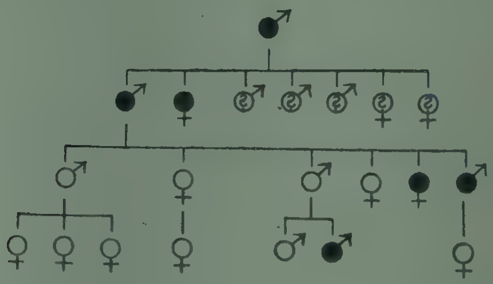

FIG. 117.

Asthma, after Ullmann.

hay fever. A genealogical tree showing the heredity of asthma is reproduced in Fig. 117.[1]

In asthmatics and in their relatives (even in those who remain free from asthma) we find an excess of a particular kind of white blood corpuscles, the so-called "eosinophil leucocytes". It would seem from this that the heredity factor which underlies asthma does not always lead to the occurrence of paroxysms. J. A. Buchanan[2] found that in 17 families in which one of the parents suffered from asthma, 8 of the children were asthmatic, and 102 were not. Other authors, such as H. Drinkwater[3] and Miss Adkinson,[4] found a much more marked accumulation of cases in particular families. We shall doubtless learn more about the heredity of this disease when the apparently healthy members of asthmatic families have had their blood examined, and when the reactions of their vegetative nervous systems have been more extensively studied. In two instances, asthma has been observed to occur under similar conditions in twins, which suggests that if asthma is to manifest itself actively there must be at work other heredity factors in addition to the specific allergic taint (polymerism). The much more frequent occurrence of asthma in males than in females may be due, either to the heredity factors being recessively sex-linked, or else to a peculiar predisposition of the male sex as such (a partial sex limitation).

Common in asthmatic families is the disorder known as *colica mucosa*, in which attacks of colic occur, attended by the evacuation of fragments or even intestinal casts of tenacious mucus. Other ailments associated with asthma are migraine, urticaria, eczema, Quincke's oedema, hay fever, and gout. The relationships are perhaps similar to those which were discussed in connexion with the diatheses of childhood (see above, p. 331).

The occurrence of *gall-stones* is likewise connected with the allergic reaction. An abnormal secretion of mucus such as that which occurs in the bronchial tubes during asthma or in the intestines during collica mucosa, may perhaps occur in the gall-bladder and there give the first impetus to the formation of gall-stones—especially if a bacterial inflammation spreads up from the intestine. There will be a deposit of cholesterin, and later of lime salts. Weitz tells me that his own clinical experience indicates that there may be a very marked family prevalence of gall-stone trouble, and that he has been led by this to suppose that the heredity is dominant in a great many cases at least. I reproduce from G. P. B. Huddy[1] a family tree showing an apparently dominant heredity. In a genealogical tree, for which I am indebted to Dr. Agnes Bluhm of Berlin, we see gall-stones and gout occurring in different members of the same family, and even in the same individuals. In our discussion of gout we have seen reason to believe that this coincidence is not an accidental one.

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[1] H. Ullmann, Zur Frage der Vererbung vegetativer Symptomencomplexe, "Deutsches Archiv für klinische Medizin", vol. CXLIV, 1924, nos. 1 and 2.

[2] Op. cit., see above, p. 353.

[3] Mendelian Heredity of Asthma, "British Medical Journal", 1909, no. 1.

[4] The Behaviour of Bronchial Asthma as an Inherited Character, "Genetics", 1920, no. 5.

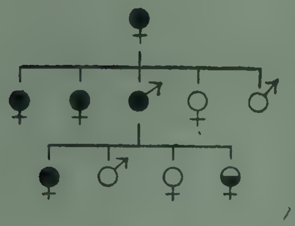

FIG. 118.

Gall-stone trouble, after Huddy.

The woman indicated by a half-black circle suffered from inflammation of the gall-bladder, but there was no evidence of the existence of a gall-stone.

In postmortem examinations we often find gall-stones in the bodies of persons who did not, so far as we know, suffer from attacks of gall-stone colic when they were alive. We are therefore compelled to suppose that gall-stones are much commoner than is indicated by the frequency of gall-stone colic or of painful inflammatory affections of the gall-bladder. This circumstance will obviously render the study of the heredity of gall-stone trouble very difficult. In the postmortem room we find gall-stones in about every tenth body,

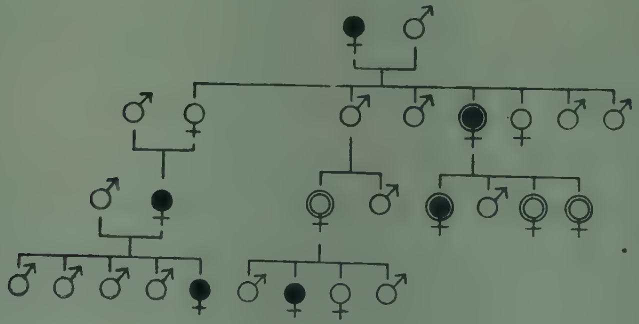

FIG. 119.

Gall-stone trouble, after Bluhm.
The circles enclosed with a double ring indicate persons suffering from gout.

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[1] A Study of the Family Histories of Three Hundred Patients suffering from Chronic Upper Abdominal Lesions, "Lancet", vol. CCIX, 1925, no. 6.

and about four times as often in women as in men. This sex proclivity may, perhaps, be connected with tight-lacing.

Chronic hereditary jaundice (constitutional haemolytic jaundice) depends upon an abnormal liability of the red blood corpuscles to break up. It would, perhaps, be better to speak of it as the haemolytic diathesis. (British and American authors sometimes speak of the disease as "cythaemolytic icterus"). The haemoglobin pours into the blood in excess from the disintegration of the corpuscles, is in the liver transformed into bile pigments, and gives the skin a jaundiced tint. Another result of the haemolysis is a high degree of anaemia. The excessive destruction of the red blood corpuscles takes place in the spleen, and the condition can be considerably improved by the surgical removal of that organ.

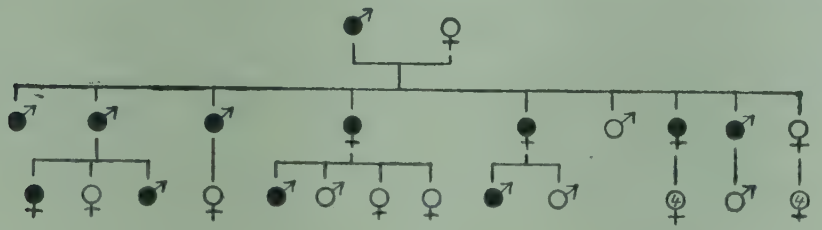

FIG. 120.

Hereditary jaundice (the haemolytic diathesis), after Gänsslen. (Children dying in infancy have been omitted.)

According to researches of M. Gänsslen,[1] the haemolytic diathesis is due to a dominant (or, perhaps, intermediate) heredity factor. In Fig. 120 I reproduce a family tree from this author. The heredity factor does not always produce the complete clinical picture of the disease. In about 40 % of the carriers, there is no obvious jaundice. Nevertheless, according to Gänsslen, even those who "exhibit no more than a trace of the disease" transmit the hereditary taint. The severity of the disease and the precise nature of the manifestation appear, in great measure, to depend upon the influence of certain other heredity factors cooperating with the specific taint which produces the haemolytic diathesis, so that the disease would seem to have a polymeric causation. A very extensive family tree compiled by Gänsslen, relating to 171 persons, shows extremely various grades of the disease.

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[1] Ueber haemolytischen Icterus, "Deutsches Archiv für klinische Medizin", vol. CXL, 1922, p. 210.

Carriers of the taint almost always have gall-stones, this being manifestly due to the formation of bile pigments in great excess. In a very large proportion of the patients, Gänsslen also found a peculiar vertical extension of the head (hypsicephaly), whose production appears to be favoured by the same hereditary taint. Gänsslen has likewise found that anomalies in almost all the organs are apt to coexist with the haemolytic diathesis. This may be supposed to be due to an indirectly determined correlation of the kind discussed above on p. 312 in connexion with the account of the stigmata of degeneration.

The transient jaundice which is fairly common in the new-born (icterus neonatorum) has a marked tendency to run in families; so has the much rarer grave form of the disease which is usually fatal. We have, however, no precise information concerning the heredity of these disorders.

In hereditary enlargement of the spleen (*splenomegaly*) during the years of growth the spleen undergoes a disproportional enlargement. At the same time, in these patients, the effect of sunlight on the skin is to tint it a brownish yellow. The enlargement of the spleen is due to the proliferation of peculiar groups of cells, which are found also in the liver, the lymphatic glands, and the bone marrow. This rare disease seems to be practically innocuous. According to family trees published by A. M. Gossage[1] and A. Plehn,[2] the heredity appears in most cases to be dominant; but in certain individuals affected with the taint it is very little, if at all, manifest, and it may therefore seem to skip one or more generations.

Cirrhosis of the liver usually originates as the outcome of toxic influences (alcohol, syphilis, malaria); but inasmuch as these poisons act on innumerable persons without producing cirrhosis of the liver, we must assume that a hereditary weakness of the organ is a necessary contributory cause—and, in fact, cirrhosis of the liver has been observed to run in families. In various organs, a premature breakdown may occur as the outcome of a weakness of the rudiment. Schuszik[3] has reported the cases of three sisters who died of cirrhosis of the liver at the ages of two and three years respectively.

[1] The Inheritance of Certain Human Abnormalities, "Quarterly Journal of Medicine", vol. I, 1908, p. 331.

[2] Familiäre Milz- und Lebervergrösserung mit Anämie und gutartigem Verlauf, "Deutsche Medizinische Wochenschrift", 1909, no. 40.

[3] Ueber einen Fall von familiärer kindlicher Leberzirrhose, "Archiv für Kinderheilkunde", 1920.

In the year 1912, Wilson described a peculiar disease to which has been given the name of *pseudosclerosis*, wherein cirrhosis of the liver is associated with focal disorders in the brain. In most instances the trouble has been observed in brothers and sisters, and its heredity would therefore seem to be recessive. About seventy cases have already been recorded, so the malady is probably not of the rarest, but has hitherto not been recognised.

The fatal disease known as progressive *pernicious anaemia* has often been observed in several members of the same family. In many cases the cause seems to be a failure of the haematopoietic (blood-forming) organs—bone marrow—which become prematurely senile owing to a hereditary weakness. In other instances, the malady seems to be the outcome of external noxious influences, such as syphilis. But even in those cases in which pernicious anaemia is caused by the toxins produced by the broad tapeworm (botriocephalus latus), hereditary predisposition would seem, as O. Schaumann,[1] professor of medicine at Helsingfors University, has insisted, to be of considerable importance. For only in exceptional cases does the botriocephalus, whose encysted form is found in fishes, lead to the onset of pernicious anaemia. Schaumann, however, had under observation twenty-four cases in which at least two members of a family were affected with botriocephalus anaemia. Mustelin[2] was able to trace the malady through three generations. He believes the heredity to be dominant.

The hereditary disorder which Meulengracht[3] speaks of as "pernicious anaemia" is not the disease usually known by that name, being what other authors term the haemolytic diathesis.

In pernicious anaemia, the stomach usually ceases to secrete hydrochloric acid (achylia gastrica), this failure of secretion being it would seem due, not simply to the anaemia, but to the working of some other still unknown cause. Persons suffering from pernicious anaemia will often be found to have relatives who suffer from achylia gastrica. The same heredity factor may be presumed to be at work in both instances.

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[1] Ueber das familiäre Auftreten der perniziösen Anämie, "Finska Läkaresällskapets Handlingar", Helsingfors, 1918.

[2] "Acta Medica Scandinavica", vol. XXXVI, no. 4.

[3] Fünf Fälle von perniziöser Anämie in derselben Familie, "Ugeskrift for Laeger", 1925, no. 25.

The mild form of anaemia which is known as *chlorosis*, a disorder of girlhood, likewise tends to run in families.[1] Chlorosis can easily be relieved by the administration of preparations of iron. Experience shows, however, that other circumstances than a lack of iron must be contributory causes of the disease. A few decades ago, this form of anaemia was extremely common in girls, but has now become rare. T. Deneke[2] considers that the only plausible explanation of this decline in the frequency of chlorosis is the discontinuance of tight-lacing. He supposes that in the days when a wasp waist was fashionable, the pressure of tight stays on the spleen gave rise to anaemia, the spleen being one of the chief organs concerned in the regulation of the supply of red blood corpuscles.

The disease known as *leukaemia*, a fatal disorder in which the blood contains a great excess of leucocytes (white blood corpuscles), is of more than one kind, according as the excess is of the myeloid leucocytes with lobate nuclei or of the lymphatic leucocytes with round nuclei. Both myeloid and lymphatic leukaemia have been found to run in families. Presumably, however, external influences are of considerable importance in its origination, just as in the case of malignant tumours, to which the leukaemias are akin.

In *polycythaemia* we have to do with a marked increase in the number of the red blood corpuscles, and apparently in the quantity of the blood as well. This rare condition is associated with certain changes in the eye. E. Engelking[3] was able to trace its inheritance through three generations. The heredity, therefore, would appear to be dominant.

Paroxysmal haemoglobinuria, in which blood pigment appears in the urine from time to time after exposure to cold (often very slight cold), depends in many cases upon hereditary predisposition. We do not know much about the causation of this disease, but it would seem probable that the diathesis may in some instances be an outcome of syphilis.

Constitutional haematuria, in which haemorrhage from the kidney leads to the appearance of blood in the urine, has also been found to run in families.

Constitutional albuminuria, the excretion of albumin in the urine without any demonstrable affection of the kidneys, has likewise been observed as a family proclivity. In families thus affected, we also find that infectious and other diseases of the kidney are prone to occur, so that there appears in these families to exist a hereditary weakness of the kidneys.

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[1] O. Naegeli, Blutkrankheiten und Blutdiagnostik, fourth edition, 1923.

[2] Ueber die auffallende Aufnahme der Chlorose, "Deutsche Medizinische Wochenschrift", 1924, no. 27.

[3] Ueber familiäre Polyzythämie, "Klinische Monatsblätter für Augenheilkunde", vol. LXIV, 1920, p. 645.

The diseases of the kidney proper are found to run in families. I have already referred to nephrosclerosis as a consequence of hereditary hypertension. Genuine *contracted kidney* is also a family disease in many instances. Volhard[1] had under observation two brothers who died of this disease in early youth. We must suppose that in these cases there was a hereditary weakness of the kidney tissue, manifesting itself in a premature senility of the cells of the organ.

The inflammation of the kidneys that results from infection (*glomerulonephritis*) is likewise to so great an extent a family disorder that we must suppose hereditary predisposition to be an important contributory cause. P. K. Pel[2] observed a family in which eighteen members died of glomerulonephritis. Dickinson,[3] too, has described eighteen cases of chronic nephritis appearing in one family during three generations. It is obvious that in such instances there must be so marked a susceptibility that the infection which acts as exciting cause must be of subsidiary importance, and hereditary influences must be decisive. In these families, the heredity was manifestly dominant. Dominant, too, would seem to have been the heredity in the family observed by W. Weitz[4] (see Fig. 121).

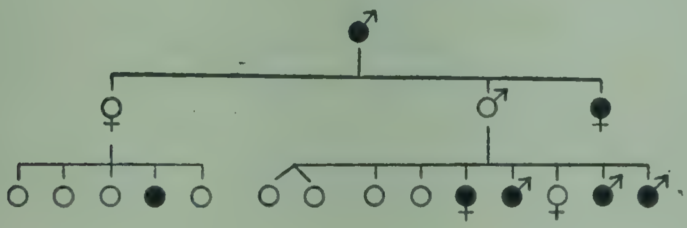

FIG. 121.

Inflammation of the kidney (glomerulonephritis), after Weitz.

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[1] Quoted by L. R. Grote, Grundlagen ärztlicher Betrachtung, Springer, Berlin, 1921.

[2] Die Erblichkeit der chronischen Nephritis, "Zeitschrift für klinische Medizin", vol. XXXVIII, 1899, p. 127.

[3] Diseases of the kidney, 1877.

[4] Die Bedeutung der Erblichkeit für die Aetiologie, "Ergebnisse der gesamten Medizin", vol. V, 1925, nos. 3 and 4.

Perhaps it is the rule for heredity to be dominant in this affection.

In many families it would seem that the kidneys are only susceptible to certain specific noxious influences. There are families in which several members have suffered from nephritis after scarlet fever, though nephritis cannot be regarded as a very frequent sequel of scarlatinal affection. Scarlatinal nephritis does not usually result in permanent injury to the kidneys.

In Fig. 122, I print the genealogical tree of a family in which all four children, then respectively 14, 12, 10, and 5 years of age, suffered simultaneously from nephritis. Only the youngest child, a girl, had the ordinary symptoms of scarlet fever, but we must suppose that the three elder children had the disease at the same time, though without the exanthem. The mother had had severe scarlet fever followed by nephritis at the age of 10, and subsequently had had nephritis again in one of her pregnancies. The father had also suffered from nephritis at the age of 11. The father and the mother were first cousins.

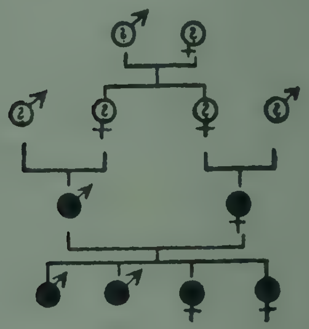

Fig. 122.

Scarlatinal nephritis (personal observation).

There is a not very rare malformation of the kidney known as *cystic kidney*, in which the renal tissue contains numerous cavities. This has also on several occasions been observed to run in families. Inasmuch as the malformation can only be detected on postmortem examination, the study of its heredity is very difficult.

Hereditary predisposition is also of great importance in connexion with the aetiology of those forms of heart failure which are sequels of *valvular inflammation of the heart*. Such valvular inflammation (endocarditis) occurs most often as a sequel of infectious articular rheumatism (polyarthritis), but may sometimes occur without any such antecedent. Articular rheumatism, like valvular inflammation of the heart and consequent heart failure, is often observed to run in families. A family tree for which I am indebted to Hanhart (Fig. 123) suggests a dominant heredity. Other authorities have likewise been able to trace valvular disease of the heart through three or more generations.[1]

In a family described by Strebel[2] and Steiger there was a remarkable association between endocarditis and dislocation of the lens (see Fig. 124, and see above, p. 242).

The family tree given in Fig. 124 suggests a sex-linked dominant heredity. This harmonises with the observation made by M. Herz[3] to the effect that "in the majority of instances it is the mother who transmits this predisposition to the children". For the rest, however, the predisposition to valvular inflammation of the heart cannot be regarded as a biological unity.

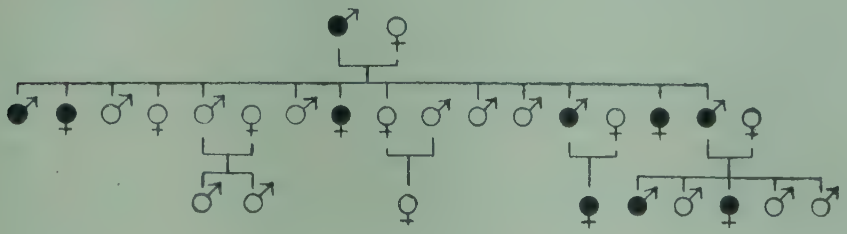

FIG. 123.

Valvular disease of the heart following upon articular rheumatism and endocarditis, after Hanhart.

In many families, so large a proportion of the members suffer from valvular inflammation of the heart that, presuming the heredity to be dominant, we have good reason to dread the onset of valvulitis in almost every case. All persons

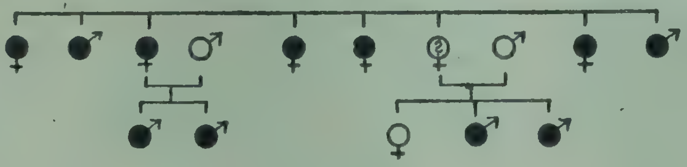

FIG. 124.

Heart failure as a sequel to endocarditis, after Strebel (extract). In the case of the woman indicated by a circle enclosing a question mark, it is doubtful whether she suffered from heart disease. All the individuals denoted by black circles suffered simultaneously from heart disease and from dislocation of the lens.

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[1] L. Mohr, Ueber familiäre Herzfehler, "Medizinische Klinik", 1905, no. 23.

[2] Korrelation der Vererbung von Augenleiden, etc., "Archiv für Rassenbiologie", vol. X, 1913, no. 4.

[3] Ueber den Einfluss der Heredität auf die Entstehung von Herzkrankheiten, "Münchener Medizinische Wochenschrift", 1912, no. 8.

susceptible to rheumatic infection seem to be exposed to it from time to time; and, on the other hand, persons who are not susceptible apparently escape infection notwithstanding reiterated exposure thereto.

Valvular disease of the heart sometimes ensues in persons who do not seem to have had antecedent valvulitis or articular rheumatism. This takes the form of mitral stenosis (narrowing of the aperture leading from the left auricle into the left ventricle), and occurs especially in women. Ferranini[1] has been able to trace mitral stenosis of this kind through several generations.

We seem to be concerned here with a congenital defect, and inasmuch as mitral stenosis appears to be about twice as common in women as in men, it is possible that in most cases the disorder depends upon sex-linked dominant heredity. Since, speaking generally, congenital valvular disorders of the heart are commoner in male children than in female, we are led to suppose that in many of these defects the heredity is recessively sex-linked. However this may be, there can be little doubt that the sex chromosome has a special part to play in the formation of the heart.

According to Grote, constitutional tachycardia (undue frequency of the pulse) often tends to run in families. Many such cases of tachycardia are presumably due to hyperthyroidism. On the other hand, there are numerous families in whose members we note a tendency to a remarkably infrequent pulse.

According to M. Herz,[2] the occurrence of nervous disorders of the heart in parents and children alike is frequently observed. There are many families in which death from heart failure consequent upon arteriosclerosis occurs in numerous individuals during the fifth and sixth decades of life (see above under hypertension, p. 349). I have already referred to heart trouble in connexion with hyperthyroidism.

There are various congenital malformations of the heart concerning whose heredity we have very little information because they are hidden disorders.[3] The commonest and least grave among these congenital defects is the persistence of a foetal aperture in the septum between the two auricles (patent foramen ovale). This condition, which is found on the postmortem table in almost every third body, is probably hereditary. The presence of an aperture in the septum between the two ventricles has been traced through several generations of a family. An abnormal communication between the pulmonary artery, which originates out of the right ventricle, and the aorta, which originates out of the left ventricle—patency of the foetal ductus Botalli—has been observed in six brothers and sisters.[1] Abnormal smallness of the pulmonary artery, interfering gravely with the blood circulation and inducing cyanosis, may be presumed in most cases to be idioplasmatically determined. An unduly vertical position of the heart and an abnormal smallness of the organ are associated with the asthenic or hypoplastic constitution, and are, like this, hereditary.

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[1] Quoted by L. Mohr, op. cit.

[2] Ueber den Einfluss der Heredität auf die Entstehung von Herzkrankheiten, "Münchener Medizinische Wochenschrift", 1912, no. 8.

[3] H. Vierordt, Die angeborenen Herzkrankheiten, Vienna, 1898.

It would seem that hereditary predisposition plays a more important part than does unsuitable feeding in the causation of *disorders of the stomach.* Weitz, in his study of twins, found a remarkable coincidence of gastric trouble in mon-oval twins. We seldom find that "errors of diet" lead to serious disturbance of health except in persons who suffer from a "weak stomach". Muscular weakness and flaccidity (atony) of the stomach occur especially in persons with an asthenic constitution. In like manner, inadequate secretion of the gastric juice (achylia) is in large measure hereditarily determined. "Nervous dyspepsia" generally manifests itself as one among many symptoms of a general anomaly of the nervous system. Excessive secretion of hydrochloric acid in the gastric juice (hyperchlorhydria) is often found to run in families, being generally the expression of undue irritability of the vegetative nervous system, of the so-called vagotonia. To the same group of disorders belongs the inclination to a convulsive closure of the pyloric outlet of the stomach (pylorospasmus), which manifests itself especially during childhood in the form of frequently recurrent and obstinate vomiting. Hyperchlorhydria favours the onset of what is known as gastric ulcer, which is produced by the self-digestion of an area of the gastric wall, attended by severe bleeding, prolonged illness, and sometimes proving fatal.

According to L. R. Grote,[2] in over 60 % of all cases of gastric ulcer we find that other members of the family have suffered from the same complaint. H. Strauss[1] found a smaller percentage, but in his series of cases likewise there was a well-marked tendency for the affection to run in families. We have to bear in mind that in the postmortem room we often discover the scars of gastric ulcers in persons who were not known during life to have suffered from the disease. Spiegel[2] has found that in the parents and in the brothers and sisters of persons who have suffered from gastric ulcer, gastric ulcer is five times as common and gastric cancer is six times as common as in the relatives of persons in whom there is no history of gastric ulcer. E. Adler's[3] investigations led him to similar results. There is unquestionably this much connexion between gastric ulcer and gastric cancer, that gastric cancer develops by preference upon the soil of an old gastric ulcer. I shall have to return to the topic of the heredity of cancer. A. Ohly[4] has published a brief report concerning nine families subject to ulcers of the stomach or duodenum. G. P. B. Huddy[5] found that when near relatives suffered from gastric ulcer or from duodenal ulcer the seat of the ulcer was generally much the same in different members of the same family.

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[1] O. de la Camp, "Die deutsche Klinik von Leyden und Klemperer", vol. IV, p. 207.

[2] Der Einfluss der Konstitution auf die Pathogenese der Magendarmerkrankungen, Marhold, Halle, 1920.

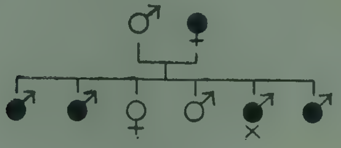

Fig. 125.

Duodenal ulcer, after Huddy.

The man indicated by a × suffered from gastric ulcer; the other individuals indicated by black circles suffered from duodenal ulcer.

Hereditary predisposition seems to be of importance in the production of *chronic constipation* or habitual constipation. The same remark applies to the (less frequent) proneness to diarrhoea, from which many persons suffer throughout life. Spastic constipation is usually one of the effects of vagotonia, whereas atonic constipation is usually one of the effects of asthenia.

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[1] Ueber hereditäres und familiäres Vorkommen von Ulcus ventriculi et duodeni, "Münchener Medizinische Wochenschrift", 1921, no. 8.

[2] Organdisposition bei Ulcus pepticum, "Deutsches Archiv für klinische Medizin", vol. CXXVI, 1918, p. 45.

[3] Ueber hereditäres Vorkommen des Magen- und Zwölffingerdarmgeschwürs, "Archiv für Verdauungskrankheiten", vol. XXXVII, 1926, p. 393.

[4] Familiäres Auftreten von Ulkus im Gastroduodenaltraktus, "Münchener Medizinische Wochenschrift", 1923, no. 37.

[5] A Study of the Family Histories of Three Hundred Patients suffering from Chronic Upper Abdominal Lesions, "Lancet", vol. CCIX, 1925, no. 6.

What is known as *Hirschsprung's disease*, in which, owing to the fact that the colon (large intestine) is of abnormally large calibre, there is from earliest childhood a tendency to the accumulation of great masses of faeces in the large gut, appears to be a hereditary disorder. In a family tree studied by Gänsslen,[1] the heredity seems to have been dominant.

Passing now to consider diseases of the air passages and the lungs, we find that chronic *bronchial catarrh* and the frequently associated *emphysema* have a notable tendency to run in families. On the other hand, a hereditary "rigid dilatation of the thorax", in which the thorax is more or less fixed in the position of extreme inspiration, seems to be a cause of chronic emphysema and chronic bronchitis.

The disease known as *ozaena*, in which offensively smelling crusts form in the nose, when not due to syphilis or some other specific infection, would appear to be hereditarily determined. The immediate local cause is an atrophic condition of the nasal mucous membrane, attended by the formation of a fluid secretion which dries into evil-smelling crusts. The trouble often runs in families.[2]

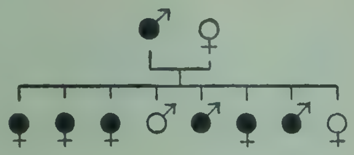
FIG. 126.

Abnormal width of the large intestine (Hirschsprung's disease), after Gänsslen.

According to Siemens, in exceptional instances ozaena is due to a recessive sex-linked heredity factor, which simultaneously produces anidrosis, hypotrichosis, and dental defects.

The question as to the hereditary predisposition to pulmonary tuberculosis and to inflammation of the lungs will be considered in the ensuing subsection.

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[1] Quoted by W. Weitz, Die Bedeutung der Erblichkeit für die Aetiologie, vol. V, 1925, nos. 3 and 4.

[2] W. Albrecht, Die Bedeutung der Keimsubstanz für die Entstehung der Ozaena, "Zeitschr. f. Hals-, Nasen- und Ohrenheilkunde", vol. XV, 1926, nos. 2 to 4.

7. SUSCEPTIBILITY TO INFECTIOUS DISEASES

Until a comparatively recent date, syphilis was regarded as a typical instance of a "hereditary disease". I need hardly tell the readers of this book that the transmission of an infectious disorder from parents to children has nothing whatever to do with the inheritance of disease in the proper sense of the term. In the case of syphilis, in particular, transmission only occurs through the mother, the infection passing by way of the maternal circulation to the developing embryo. There is no such thing as the transmission of syphilis from father to child without the mother being infected.

When we are discussing heredity as a contributory factor in the causation of infectious diseases, we can only be concerned with susceptibility to the exciting causes of infectious disease.

Every infectious disorder represents a struggle between the invading microscopic germs and the infected organism. Speaking generally we may say that each species of animal has its own peculiar kind of microscopic parasites. Man is immune to most of the infectious diseases from which domesticated animals suffer, and conversely, domesticated animals are for the most part immune to the infectious diseases from which man suffers. But, apart from this, human beings vary to a considerable extent in their susceptibility to the infectious disorders affecting mankind. When, for instance, diphtheria bacilli gain access to the mucous membrane of a child's throat, that child will not necessarily suffer from diphtheria. There are, in fact, three grades of susceptibility. In some children, the bacilli cannot gain a lodgment at all; in other children, they gain a lodgment and multiply, but the hosts do not become obviously ill; in children of the third category, the illness we know as diphtheria develops. Once more, among those who actually suffer from diphtheria, the severity of the disease varies greatly from person to person. Some children have no more than a moderately sore throat; and at the other end of the scale are the children who die from the disease. In the case of many infectious disorders, such as measles, smallpox, and influenza, most human beings who have not already had the disease are susceptible, but we nevertheless find that there are certain persons who remain free from illness notwithstanding repeated exposure to infection. There are yet other diseases, such as cerebrospinal meningitis and scarlet fever, from which only a minority of those exposed to infection suffer.

An individual's susceptibility to a disease is also spoken of as his *predisposition.* Non-susceptibility or the lack of predisposition to an infection is termed *immunity*. Thus immunity and predisposition are inverse variables.

Hitherto it has been usual to distinguish between congenital and acquired immunity or predisposition, as the case may be. The distinction, however, is not wholly tenable. What is called congenital immunity is partly acquired (in the mother's womb). On the other hand, acquired immunity is potentially congenital, seeing that its foundations must exist in the hereditary equipment. The best way, therefore, will be to draw our distinction between hereditary (*idiotypical*) and non-hereditary (*paratypical*) immunity or predisposition. Persons of like idiotypical constitutions will, owing to differences in the environmental influences that have acted upon them, display different degrees of susceptibility to various diseases, and these differences are exclusively paratypical. If, on the other hand, it were possible for a number of persons from birth onwards to be exposed to identical environmental influences, their respective predispositions to disease would nevertheless not be identical, for there would be variations dependent upon their different hereditary equipment; these differences would be exclusively idiotypical. The actual (phenotypical) predisposition of an individual is always jointly determined by his hereditary equipment and by the environmental influences which have acted upon him; and when we speak of differences within the sphere of one set of determinants, it is always more or less on the proviso that the other set of determinants are to be considered as having acted uniformly. Two individuals could only have perfectly identical susceptibility to illnesses if they had (like mon-oval twins) identical hereditary equipments and had ever since birth been exposed to precisely identical environmental conditions.

The normal organism has at its disposal a large number of protective institutions to safeguard it against the exciting causes of disease. The normal skin when uninjured protects against the invasion of the body by the exciting causes of wound infection. The ciliary movement of the cells lining the respiratory mucous membrane is able to sweep away the germs of disease before they can become lodged in the tissues. The hydrochloric acid in the normal gastric juice is able to render harmless cholera bacilli and other disease-producing germs that have been taken into the stomach with food or drink. Pathogenic microbes which have found their way in through wounds can be caught and destroyed in the lymphatic glands. Even after pathogenic organisms have undergone multiplication within the body, the organism has numerous weapons of defence against the invaders. The white blood corpuscles, especially those with lobate nuclei, are able to ingest and destroy bacteria and other micro-organisms, or to encyst them and render them harmless. A great many infections are counteracted by a vigorous proliferation of these phagocytes (eating cells). The white blood corpuscles and doubtless some of the other body cells also produce chemical protective substances to resist micro-organisms and their toxins. Such protective substances (antitoxins) are also formed in restricted quantities in the absence of specific simuli and simply as the outcome of hereditary predisposition. But when the body is invaded by the exciting causes of disease, there ensues a marked increase in the protective substances. What usually happens is that invasion by specific organisms leads to the formation of specific antitoxins to counteract these particular kinds of microscopic invaders. Inasmuch as many of the antitoxic or protective substances persist within the body for a long time after the illness has been overcome, or else because owing to the changes induced in the tissues by the illness the formation of antitoxins is continued for a lengthy period, the body is for a longer or shorter time protected against this particular disease. That explains the production of what is known as acquired immunity which, thus, so far as it is acquired, is paratypical.

But the capacity to form antitoxins is, in fact, idiotypically determined. Hereditary, likewise, is the capacity to form a specific antitoxin as the need for this or that antitoxin may arise; and the sum total of these capacities varies much from person to person. We cannot, as L. Hirszfeld[1] declares we can, regard the organism as a serological tabula rasa upon which the stimuli to immunisation can write anything they please. On the contrary, the formation of immunising substances must be regarded as nothing more than a development and intensification of capacities pre-existing as parts of the hereditary equipment. Inasmuch as the struggle against pathogenic micro-organisms is for all of us a matter of life or death, we must look upon these numerous possibilities of reaction in the way of the formation of antitoxins as having been fostered in us by natural selection.

A. C. Hagedoorn, La Brand, and A. L. Hagedoorn,[2] in the course of breeding experiments on mice, have noted that the immunity towards certain pyogenic organisms (staphylococci) has a simple dominant heredity; but the outcome of their experiments might be just as well summarised by saying that the susceptibility to staphylococcal infection has a simple recessive heredity. We may presume that other specific susceptibilities depending upon the lack of certain normally present specific means of defence will as a rule have a recessive heredity.

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[1] Krankheitsdisposition und Gruppenzugehörigkeit, "Klinische Wochenschrift", 1924, no. 46.

[2] Inherited Predisposition for a Bacterial Disease, "American Naturalist", vol. LIV, 1920, p. 368.

Seeing that an active adaptation cannot take place to a degree exceeding that provided for by the hereditary equipment, it follows that acquired immunity cannot as such be transmitted by inheritance. Of course an immunity due to the presence of active and freshly formed antitoxins in the mother's blood can be transmitted to the embryo through the placental circulation, or to the nursling through the milk, but this process is not one of inheritance; and there is no corresponding physiological mechanism by which an acquired immunity in the father can be transmitted to his offspring.[1] Owing, however, to the transmission of immunity from the mother, most children are immune to such infectious diseases as measles during the first year of life, and especially during the first few months. Many physicians and hygienists are inclined to assume, more or less unconsciously, that the immunisation of a race is possible along these lines, but such a supposition is erroneous. The possibilities of reaction dependent upon the hereditary equipment are quite uninfluenced by the extent to which these possibilities are utilised within the lifetime of the individual.

Nevertheless it cannot be regarded as of no moment to the hereditary equipment of a race whether the possibilities of reaction based upon that equipment are or are not utilised, seeing that the effects of selection have to be taken into account. It is by the elimination of predisposed families and by the survival of those which are comparatively immune—and not by a supposed "inheritance of acquired characters"—that we must explain the lower susceptibility of negroes to malaria and yellow fever, the lower susceptibility of the natives of Hindustan to cholera, and the lower susceptibility of Europeans to tuberculosis. Negroes and the natives of Hindustan, in whose homeland tuberculosis plays no part, and who therefore have not been selected to resist tuberculosis, are much more susceptible to this disease than are Europeans. Two pioneer works on this subject of the acquirement (through selection) of immunity to infectious diseases are G. Archdall Reid's Present Evolution of Man, 1896, and Hugo Ribbert's Die Bedeutung der Krankheit für die Entwicklung der Menschheit, Bonn, 1912 (English translation by E. and C. Paul, Heredity, Disease, and Human Evolution, New York, 1918).—Sir G. Archdall Reid is the author of other notable works on Heredity. —Concerning tuberculosis, see J. J. Vassal, La tuberculose chez les peuples primitifs, "Revue de Physiologie", Paris, vol. X, No. 1, February, 1929.

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[1] L. Hirszfeld and J. Seydel, Untersuchungen über Vererbung der Antikörper II. Ueber die Giftempfindlichkeit der Nachkommen immunisierter Väter, "Zeitschr. f. Hygiene u. Infektionskrankheiten", vol. CIV, 1925, no. 3.

L. T. Webster[1] has shown in experiments on animals how selection. is effected by immunisation. He infected a number of mice with mouse typhoid. In about 70 % of the animals the infection proved fatal. The offspring of the surviving mice were in turn infected, and of these only 42 % succumbed to the disease. In the third generation, the mortality resulting from infection was only 15 %. Webster inferred from this that in these mice the power of resistance was to a great extent determined by hereditary predisposition, and (generalising the notion) that the power of resistance may be increased in any population by selection. The offspring of a strain of mice which had survived two typhoid infections and a sublimate intoxication (the survivors were only a small minority) had a typhoid mortality of 24 %, whereas in control experiments the mortality was 70 %. It is, moreover, noteworthy that the offspring of mice which had survived typhoid infection had a markedly increased power of resistance to sublimate poisoning as well. Thus selection by this infectious disorder resulted, not only in an increase in the power of defence against that specific infection, but also in a general increase in vital tenacity.

Furthermore, an individual's liability to an infectious disorder is not exclusively dependent upon that individual's constitution, for the vigour of the exciting cause of the disease is a co-determinant.

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[1] Microbiotic Virulence and Host Susceptibility in Paratyphoid-Enteritidis Infection of White Mice, IV, The effect of Selective Breeding on Host Resistance, "Journal of Experimental Medicine", vol. XXXIX, 1924, p. 879.

The idiotypical constitution (the race) of the exciting organism plays a very important part. Inasmuch, however, as this book is exclusively concerned with human heredity, the constitutional peculiarities of the exciting causes of disease need not here be further considered.

Attacks of *scarlet fever* tend very markedly to run in families—not only during a particular epidemic, which might be fully explained as the outcome of simultaneous infection, but also when longer periods than those of one epidemic are under observation. On page 372 we have already considered family tendencies to inflammation of the kidney after scarlet fever. I am indebted to Dr. Agnes Bluhm for the family tree shown in Fig. 127, wherein individuals who as children had scarlet fever twice are shown by black circles and those who had scarlet fever once are shown by half-black circles. It will be seen that there were three persons in whom the immunity produced by an attack of scarlet fever was insufficient to prevent a second attack.

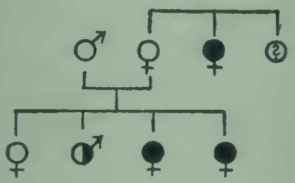

FIG. 127.
Defective formation of scarlatinal antitoxin.

It is very exceptional for any one to have *measles* more than once. There are, however, certain families whose members appear to be unable to acquire a permanent immunity to this disease, so that they may have repeated attacks.[1] On the other hand, there are persons who never suffer from measles, and we may suppose that in great measure they escape because of a hereditary immunity. When measles was first introduced by Europeans into the South Sea Islands, it was a deadly pestilence, because, in this remote part of the world, there had hitherto been no selection by measles. In the Fiji Islands during the year 1876 forty thousand among the one hundred and fifty thousand indigens died from measles.

The power of resisting *malarial infection* seems, in part, to be directly connected with the hereditary richness of the skin in pigment, just as the immunity of black swine to buckwheat disease is so determined. Concerning the Levantines,

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[1] Mathilde Salzmann, Ueber wiederholte Masern, "Zeitschr. für Kinderheilkunde", 1920.

F. Luschan reports that, in conjunction with their dark skins, an almost complete immunity to malaria has been produced by selection.[1]

As regards the *infectious catarrhs*, which usually tend to appear epidemically once or twice a year, we find that the members of some families suffer severely, whereas the members of other families suffer exceptionally and slightly. Among isolated peoples living in a comparatively wild state, what with us is the "common cold", when introduced from without, sometimes leads to severe epidemics. For instance, K. Sapper[2] reports this of the Indians of Central America.[3]

Susceptibility to catarrh seems to be in some degree connected with the inflammatory and the lymphatic diatheses (see above, p. 326 and p. 328). Most of the laity are inclined to believe that a cold in the head is due to "chill"; and many doctors still favour this view. For two decades I have directed special attention to the point, and, having observed the outbreak of dozens of colds in myself and hundreds in other persons, I have been unable to find any sound reason for believing that "chill" plays an important part in the origination of colds. A cold in the head has the typical course of an infectious disorder. Starting from a primarily infected area (usually the nasal passages close to the nostrils or else the tonsils) the inflammation spreads over the mucous membrane of the respiratory passages. When the cold is over, for a restricted period (several months, as a rule) the patient remains immune to further infection with a cold. Arctic explorers, who are exposed to very severe cold, tell us that during their travels they never suffer from colds, but are attacked as soon as they return to civilisation and come into contact with other persons. In Greenland, epidemics of colds occur when ships call. If we could all live as hermits, we should none of us suffer from colds.

A hereditary susceptibility to catarrhs not only impairs the sense of wellbeing while greatly reducing the functional capacity, but also in quite a number of persons is a contributory cause of death.

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[1] Völker, Rassen, Sprachen, Berlin, 1922, p. 69.

[2] Die Bedrohung des Bestandes der Naturvölker und die Vernichtung ihrer Eigenart, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XII, 1917, no. 2.

[3] Of the same character is the famous "St. Kilda cold". In this remote island of the Outer Hebrides (now uninhabited) the whole population used to suffer from epidemic coryza when, at long intervals, a ship called.—Translators' Note.

Some of the individuals thus predisposed perish of bronchopneumonia in infancy. During middle life, infectious catarrhs are less important as a cause of death, but cannot be ignored. In old age, once more, a large proportion of deaths are the outcome of infectious catarrhs.

We learn from Crampton[1] that, during an influenza epidemic in the years 1917 and 1918 in the Society Islands, from 15 to 25 % of all the natives died of the disease, whilst the proportion of deaths among the half-breeds was much smaller, and among persons of pure European blood the deaths were very few in number.

The susceptibility to *tonsillitis* tends markedly to run in families, and appears to be connected with the lymphatic diathesis. The same remark applies to the liability to *appendicitis*, which seems in many cases to be an infectious disorder, caused by germs akin to and at times even identical with those which cause tonsillitis.

In his study of twins, Weitz has been able to establish the hereditary character of the susceptibility to tonsillitis. In many families there is a notable tendency for tonsillitis to lead to the formation of tonsillar abscess (quinsy).

The way in which *acute articular rheumatism* and *valvular endocarditis* tend to run in families, shows that the predisposition to these disorders is hereditary. Pribram[2] has published several family trees in which articular rheumatism and endocarditis can be traced through four or more generations. R. Löwy and G. Stein[3] have found that in about half the sufferers from articular rheumatism and endocarditis the parents and the brothers and sisters of the patient have had the same diseases. In most instances, those thus affected were characterised by general constitutional weakness (see above, p. 372).

The degree of susceptibility to *diphtheria* or the existence of immunity to the disease can in some measure be ascertained by what is known as Schick's test. A small dose of diphtheritic toxin is subcutaneously injected. In susceptible persons an inflammatory reaction (redness) ensues at the site of injection, whereas in immune persons there is no such reaction. Hirszfeld has published some small family trees which appear to show that the reaction to Schick's test is determined by hereditary predisposition.[1]

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[1] On the Differential Effects of the Influenza Epidemic among Native Peoples of the Pacific Islands, "Science", vol. LV, 1922.

[2] Der akute Gelenkrheumatismus, Vienna, 1899.

[3] Zur Aetiologie des akuten Gelenkrheumatismus, "Zeitschrift für Konstitutionslehre", 1921.

Hirszfeld also believes that he has found evidence of a coupling with a particular "blood group" (see above, p. 163). The number of his observations is, however, so small that the coincidence he has observed may well have been due to chance. Apriori we have no reason to expect a coupling between hereditary trends and particular antitoxins, or between hereditary trends and particular isoagglutinins; and even if there should be a true coupling between human hereditary trends and other characters, the demonstration of its existence would be extremely difficult. At the moment, however, "blood groups", on the one hand, and couplings on the other, are in fashion. We need not be surprised, therefore, to find that the two should be conjoined. It is likely enough that in the near future confirmation of Hirszfeld's discoveries will be reported from other quarters.

A more general susceptibility to various infectious disorders may result from all kinds of constitutional anomalies. Children affected with morbid diatheses suffer more severely than others from the so-called diseases of childhood (measles, scarlet fever, diphtheria, whooping cough).[2] In cretinism, which is due to a hereditary lack of the thyroid, there is an exceptionally high susceptibility to infectious disorders. The function of the thyroid is to regulate metabolism. This gland is obviously concerned both with the origination of the febrile reaction to microbic infection (a reaction which is to be regarded as a curative effort on the part of the organism) and with the formation of antitoxin.

Important as the constitution is as regards the onset of infectious disorders, it is even more important as regards their result. Persons who, owing to hereditary determinants, suffer from heart weakness, anaemia, or defective nutrition, are apt to die of acute infectious disorders which do not seriously threaten the lives of normal persons.

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[1] H. Hirszfeld, L. Hirszfeld, and H. Brokman, Untersuchungen über Vererbung der Disposition bei Infektionskrankheiten, speziell bei Diphtherie, "Klinische Wochenschr.", 1924, no. 29.

[2] A. Czerny, Die Bedeutung der Konstitution für die Klinik der kindlichen Infektionskrankheiten, "Zeitschrift für ärztliche Fortbildung", 1913, no. 24.

Of very great importance is the question of the hereditary susceptibility to *tuberculosis*, the disease which next to syphilis brings more suffering to mankind than any other infectious disease. Although the mortality from tuberculosis in central Europe has been greatly reduced of late years, in this part of the world from 10 to 15 % of all deaths are due to tuberculosis. While it is indisputable that there can be no true tuberculosis in the absence of infection with the tubercle bacillus, there is good reason to suppose that for practical purposes hereditary susceptibility is more important than infection. Among our population most persons—in large towns at any rate—are already infected with tubercle bacilli during childhood, and nevertheless no more than a fraction of them die subsequently of pulmonary tuberculosis (phthisis or consumption). It is, indeed, true that environmental noxious influences other than the tubercle bacillus play a considerable part in causing pulmonary consumption. Above all, poor nutrition and occupational conditions (especially the inhalation of certain kinds of dust) are momentous contributory causes.

Nevertheless, heredity is one of the co-determinants of consumption. The observations made by A. Riffel[1] on numerous families are still significant to-day. W. Weinberg,[2] examining the results of the marriage of four thousand tuberculous persons, was not only able to show how markedly tuberculosis tends to run in families, but was also able to prove that among the adult brothers and sisters of tubercular patients the mortality from tuberculosis was from 168 to 180 % more than the mortality from tuberculosis among the adult brothers-in-law and sisters-in-law of the same tubercular patients; and that when a distinction was drawn between those who were more well-to-do, and those who were less well-to-do, the preponderance of the death rate from tubercle was 204 % among those in the former category as compared with 135 % among those in the latter category. The comparatively well-to-do suffered more than the others. This is a plain indication of the importance of hereditary predisposition.

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[1] Die Erblichkeit der Schwindsucht, Carlsruhe, 1902.

[2] Die familiäre Belastung der Tuberkulösen, "Beiträge zur Klinik der Tuberkulose", vol. VII, 1907; Die Kinder der Tuberkulosen, Leipzig, 1913.

B. Stiller writes in his book on asthenia:[1] "In my long experience I have known a considerable number of families in which one of the parents died of pulmonary tuberculosis, and in which the children seemed for a time to have vigorous constitutions. But in the third decade of life, in the prime, long after they had left their homes and had been removed from the possibility of domestic infection, one after another of them became tubercular and died of galloping consumption." My own experience in several families enables me to confirm this observation. Even though heredity may not have very much to do with the general liability to tubercular infection, it certainly plays a great part in favouring or not favouring the development of consumption.

Turban, a noted specialist, has found that the seat of tubercular lung disease (whether it is in the right or the left lung) is in about 80 % of the cases identical in parents and children or in brothers and sisters suffering from the disease.[2] This observation has been confirmed by others. Brehmer[3] has also observed that in the members of a particular family tuberculosis tends to originate at the same age. It has also been noticed that tuberculosis of other organs than the lungs tends in the members of any one family to attack some particular organ or group of organs (the bones, the kidneys, the cerebral membranes, as the case may be). J. Bauer[4] quotes from Kretschmer the description of twin sisters both of whom became affected with renal tuberculosis at the age of fourteen. We have good reason to expect that the further study of twins will soon provide such striking evidence of the existence of a hereditary susceptibility to tuberculosis that the matter will no longer be in dispute. At present, however, most medical practitioners doubt or even deny the existence of such a susceptibility. The reason for this scepticism is that whereas most doctors already have very clear ideas concerning infection, they have not as yet any such clear ideas concerning heredity, which for many of them still has a somewhat "mystical" flavour. This difficulty will be overcome in course of time, as biological knowledge becomes more generally diffused. We are indebted to E. Haecker for a valuable critical study of the heredity of tuberculosis.[1]

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[1] Die asthenische Konstitutionskrankheit, Stuttgart, 1907.

[2] "Zeitschrift für Tuberkulose", vol. I, 1900.

[3] Quoted by J. Bauer, Konstitution und Tuberkulose, "Zeitschrift für Tuberkulose", vol. XXXIV, 1921.

[4] Die Erbanlagen des Kindes in bezug auf Tuberkulose, "Beiträge zur Klinik der Tuberkulose", vol. LIX, 1924, no. 3.

It is plain that the hereditary susceptibility to tuberculosis cannot be regarded as a unified character; but of the various morbific heredity factors which are at work here, it is only concerning a few that we have fairly precise information. We have to thank R. Schlüter for a valuable study bearing on this problem.[2] There can be no doubt that the asthenic constitution entails an enhanced susceptibility to pulmonary tuberculosis. The experience of life-assurance companies has shown that persons who were accepted as free from tuberculosis but ultimately died from that disease had had a comparatively small chest measurement at the time of acceptance; and G. Florschütz[3] (an authority on life-insurance questions) believes that the low-grade constitution is to be regarded as a contributory cause of tuberculosis rather than as a consequence of that disease. T. Brugsch,[4] studying 226 adult tubercular patients, found that the liability of narrow-chested persons to consumption is several times as great as the liability of persons with chests of a medium girth. Only 15 % of the narrow-chested consumptives had had symptoms of that disease before the age of twenty. "This indicates that the narrowness of the chest is an antecedent to the appearance of the symptoms of pulmonary tuberculosis."

On the other hand the figures as regards the incidence of pulmonary consumption are less favourable in persons with an above-average chest measurement than in persons with an average chest measurement, this suggesting that a disproportionally large chest measurement is in some way connected with an increased hereditary susceptibility to tuberculosis. People who are discussing the connexion between consumption and the habit of body are apt to suppose that all consumptives must have a particular bodily type, or that, conversely, all persons having a "phthinoid constitution" will become victims of consumption. There is no fixed connexion of this kind, for the liability to tuberculosis is determined by a number of other heredity factors in addition to the heredity factors which determine the habit of body.

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[1] Vererbungsgeschichtliche Probleme der sozialen und Rassenhygiene, in Gottstein, Schlossmann, and Teleky's Handbuch der sozialen Hygiene, vol. I, Berlin, 1925, pp. 233 et seq.

[2] Die Anlage zur Tuberkulose, Vienna, 1905.

[3] Allgemeine Versicherungsmedizin, Berlin, 1914.

[4] Allgemeine Prognostik, second edition, Berlin, 1922.

S. Wright and P. R. Lewis[1] infected with tuberculosis about one hundred guineapigs belonging to four different inbred lines. One of these lines or stocks showed itself more resistent to tuberculosis than the others, for the animals belonging to this line lived on the average one sixth longer than those belonging to the other lines. These authorities infer that specific heredity factors must be of importance here, and they believe that the results of their experiments in the crossing of the lines justify the inference that a capacity for resistance is in general dominant, and a high susceptibility recessive. In conformity with these results, A. Govaerts,[2] as an outcome of the study of 214 families, came to the conclusion that the various human biotypes are endowed with a varying power of resisting tuberculosis.

The importance of under-nutrition in the causation of pulmonary tuberculosis is generally admitted. This implies that all the heredity factors tending to promote disturbances of nutrition and poverty of blood must indirectly increase the susceptibility to tuberculosis. In diabetes, for instance, consumption often supervenes, the tissues that contain an excess of sugar being an exceptionally favourable culture medium for the tubercle bacilli. Thus a hereditary tendency to diabetes is at the same time a hereditary tendency to tuberculosis. We see, therefore, that the heredity factors of tuberculosis are multiple. My impression is that in many cases there occurs a break-down of certain defences, this break-down taking place in some families in youth, in others in middle age, and in yet others in old age. If that be true, we have to do with a hereditarily determined premature ageing of certain organs or functions, such as we already know to exist as a factor of various other hereditary diseases (for instance, cataract, diabetes, muscular atrophy).

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[1] Factors in the Resistance of Guineapigs to Tuberculosis, with especial regard to Inbreeding and Heredity, "American Naturalist", vol. LV, 1921, no. 636.

[2] The Hereditary Factor in the Etiology of Tuberculosis, "Eugenics Record Office Bulletin", 1922, no. 23.

Altstädt[1] has also found that various members of the same family tend very markedly to react in the same way to the tuberculin test. When in persons with active tuberculosis a very small quantity of tuberculin (the toxin produced by the tubercle bacilli) is injected beneath the skin, they react by a considerable local reddening. (Pirquet's tuberculin anti-reaction.) On the other hand, a slight positive reaction signifies, not that there has been actual infection with tubercle bacilli, but that the person concerned is by his hereditary constitution more than usually susceptible to tuberculosis—just as the Schick reaction indicates an analogous constitutional susceptibility to diphtheria. Now it is noteworthy that the tuberculin reaction is predominantly negative in a large proportion of those children who are repeatedly exposed to infection because their parents suffer from bacillus-discharging forms of tuberculosis.[2]

The fact that a great many children remain immune from consumption despite extensive opportunities for infection and extremely unfavourable conditions of life suffices to show that heredity must be of importance in the production of consumption, even though the tendency of consumption to run in families can certainly be explained by other mechanisms than that of inheritance. Our net conclusion is that we no longer have to ask whether hereditary susceptibility plays a part in the origination of phthisis, but only how such susceptibility acts in particular cases, and what importance it has as compared with external noxious influences. Probably in our own population the importance of heredity outweighs that of infection, and is at least as great as that of under-nutrition and that of the inhalation of dust.

8. CANCER AND OTHER MALIGNANT TUMOURS

Malignant tumours are formed by the abnormal proliferation of cells actually belonging to the body. These cells invade the healthy tissues, and ultimately cause so much injury as to bring about death. We must suppose that a malignant tumour such as a cancer usually originates from one single morbidly modified cell, so that all the millions of cells out of which a cancerous growth is composed are produced by the division and redivision of the original cancerous cell. This growth of cancer cells is distinguished from the growth of normal cells inasmuch as it is not adapted to the structural plan of the body as a whole but proceeds regardless of the body's need for self-preservation. Thus in relation to the organised cell complex or cell community out of which they originate, cancer cells behave like parasites multiplying within the community at the community's expense. Such rapid cell growth and repeated cell division as occur in a malignant tumour occur normally in the organism only during the first stages of its development from the ovum. Towards the mucous membrane of the maternal uterus in which it has become implanted, the embryo behaves, to begin with, much like a malignant tumour. Subsequent to the early period of embryonic life, in human beings some only of the tissues retain a capacity for vigorous cell multiplication, as we see, for instance, in the multiplication of the cells of the epidermis and the connective tissue which is essential to the healing and cicatrisation of a wound. Such regenerative processes, just like those of normal growth during the developmental phase of life, are restricted in accordance with the general structural plan of the organism. Thus normal growth and regeneration are adaptive processes, whereas in the growth of malignant tumours this adaptive character is lacking. A malignant growth is morbid because it conflicts with the organism's need for self-preservation.

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[1] Immunbiologische Untersuchungen über Tuberkulosedisposition, "Beiträge zur Klinik der Tuberkulose", vol. XXXIX, 1918.

[2] Falk, "Beiträge zur Klinik der Tuberkulose", vol. LVII, 1924.

Malignant tumours are of various kinds, their classification being determined by the nature of the cells out of which they have originated and whose characters they retain more or less. The tumours originating out of epithelial cells (the covering cells of the skin and the mucous membranes, and the cells of those glands which were originally formed by the ingrowth of skin or mucous membrane) are spoken of as *cancers* or carcinomata; they are the commonest of the malignant tumours. But malignant tumours can also be formed out of connective-tissue, cartilage, bone, muscle, and nerve cells. The tumours originating out of connective-tissue cells are spoken of as sarcomata. It should be noted that malignant tumours may arise, not only out of cells which are already the outcome of a disorderly development (such malformations as naevi, for instance) but also out of cells that have hitherto been normally placed and have developed in the customary manner. [The distinction between cancers or carcinomata and sarcomata, is strictly observed in scientific medicine, but in popular parlance the term "cancer" is apt to be applied to all malignant tumours.]

Causes acting from without often contribute to the origination of a malignant tumour, although in the last analysis this is due to the transformation of a normal tissue cell into a cancer cell. The contributory effect of external causes is especially manifest in the case of certain tumours of the bladder (carcinomata and papillomata), which arise in persons exposed to the fumes of anilin, benzidin, and similar substances. In certain chemical industries it has been observed that in course of time the majority of the workers become thus affected.[1] It is noteworthy that the same noxious influence is thus competent to produce different kinds of tumour of the bladder, non-malignant or malignant as the case may be. In accordance with the method discovered by Ishikawa and Yamagiva, we can produce cancer in the rabbit by repeated painting of the skin with tar. Cancer can likewise be produced almost without fail by the long-continued effects of Röntgen rays on the skin. A. Beck[2] has also reported three cases of sarcoma which resulted from exposure to Röntgen rays, and we may presume that such cases are much commoner than has hitherto been supposed. Quite a number of the doctors and operatives who have handled Röntgen rays for long periods have perished from cancer. Chemists, too, have a disproportionally high mortality from cancer. When we further take into account the prevalence of cancer of the lips in pipe smokers, that of cancer of the scrotum in chimneysweeps, together with the cancer to which paraffin workers are subject and the frequence of oesophageal cancer in spirit drinkers, it becomes impossible to doubt the importance of external causes in the origination of malignant tumours.

Various investigators are still engaged upon the search for exciting causes of cancer. Fibiger has been able to produce cancer in rats and mice by infection with certain worm larvae. In human beings, cancerous tumours often develop as a sequel of syphilitic ulcers or scars. But it would unquestionably be wrong to suppose that cancer is an infectious disorder in the sense that it is usually

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[1] M. Nassauer, Ueber bösartige Blasengeschwülste bei Arbeitern der organisch-chemischen Grossindustrie, Wiesbaden, 1919.

[2] Zur Frage des Röntgensarkoms, zugleich ein Beitrag zur Pathogenese des Sarkoms, "Münchener Medizinische Wochenschrift", 1922, no. 17.

(or, as some have supposed, exclusively) the upshot of a definite infection from without. The communicability of certain cases of cancer depends upon the transference of cancer cells, which proliferate in the new organism. But the transformation of a cell in such a way that it becomes capable of unrestricted multiplication, or, perhaps, the removal of certain normal hindrances to such multiplication may beyond question be the result of purely inorganic influences.

In many cases, on the other hand, an inherited predisposition to the growth of malignant tumours is of decisive importance. Particularly striking is the tendency of *cancer of the stomach* to run in families. In Figs. 128 and 129 I reproduce two family trees published by J. Paulsen[1] which indicate this clearly.

As regards the family tree shown in Fig. 128, the reader should note that the man who died at twenty-eight died of accident, so that we may presume that if he had lived longer he would ultimately have suffered from cancer of the stomach. His brother who died at the age of sixty-three was still healthy when the second edition of this book was published, but since then he has perished of gastric cancer, just as did both his parents, his four elder brothers, and his sister. In the family shown in Fig. 129, the eldest son died at the age of fifty of what was described as cancer of the liver, but inasmuch as primary cancer of the liver is practically unknown we can have little doubt that he also had cancer of the stomach. It is likely enough, too, that the father of this family, who is said to have died of "obstruction of the bowel", really died of cancer of the stomach.

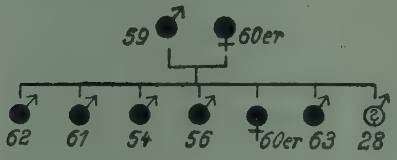

FIG. 128.
Cancer of the stomach, after Paulsen.

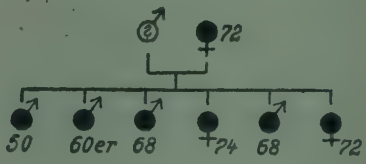

FIG. 129.
Cancer of the stomach, after Paulsen.

Since about 10% of all persons who survive childhood ultimately die of malignant tumours, among which cancer of the stomach is the commonest, we should naturally expect to find, in accordance with the doctrine of chances, an occasional run of cases in particular families. But when there is such a run of cases in a single family as is shown in one after another of the genealogical trees published here, we can no longer doubt that heredity must play its part in the origination of cancer.

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[1] Konstitution und Krebs, "Zeitschrift für Krebsforschung", vol. XXI, 1924, no. 2.

L. R. Grote[1] has reported on a family in which the inheritance of cancer of the stomach could be traced uninterruptedly through four generations. (See Fig. 130.) Such families are by no means rare. As the outcome of an enquiry addressed to the medical practitioners of Austria,[2] information was received concerning fifteen families in which cancer could be traced through four generations. There were also reports on families in which six, seven, and nine brothers and sisters had died of cancer; in thirty-four out of ninety-two cancer families there had been instances in which all the brothers and sisters of one generation perished from the disease. In the Bonaparte family, Napoleon I, three among his brothers and sisters, and his father all died of cancer of the stomach.

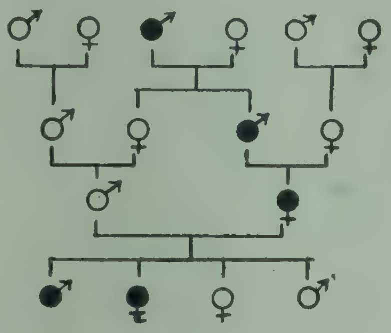

FIG. 130.

Cancer of the stomach, after Grote. (This is the same family as the one whose tree has already been given in Fig. 44, p. 243 to illustrate the heredity of cataract.)

Cancer of the stomach develops by preference upon the soil of a chronic ulcer of the stomach (see above, p. 376). In part, therefore, at any rate, the hereditary predisposition is identical with that of gastric ulcer.

J. Bauer[3] contends that cancer of the stomach arises when a general hereditary predisposition to malignant tumour (a predisposition which he regards as recessive) coincides with a particular organic disposition which is also as a rule recessive. He bases this view upon the extensive breeding experiments of Maud Slye,[4] who during thirteen years bred forty thousand mice, five thousand of

[1] Grundagen ärztlicher Betrachtung, Berlin, 1921.

[2] S. Peller, Die Ergebnisse der von der Oesterreichischen Gesellschaft für Erforschung und Bekämpfung der Krebskrankheit veranstalteten Sammelforschung, "Wiener Klinische Wochenschrift", 1922, nos. 6–8.

[3] Das Wesen der vererbbaren Krebsdisposition, "Zeitschrift für Konstitutionslehre", vol. XI, 1925, nos. 2–5.

[4] "Journal of Cancer Research", vol. VII, 1922, p. 107.

which became affected with cancerous tumours. In some of the lines almost all the mice became affected with cancer. Mice belonging to the F_1 generation of the crossing with cancer-free lines remained healthy, but in the F_2 generation cancer recurred. We see, then, that the cancer factor in these mice was recessive. It would, however, be premature to infer from these experiments that all the heredity factors capable of originating a predisposition to cancer must be recessive. Our experience of the heredity of cancer of the stomach in human beings suggests rather that the taint is dominant.

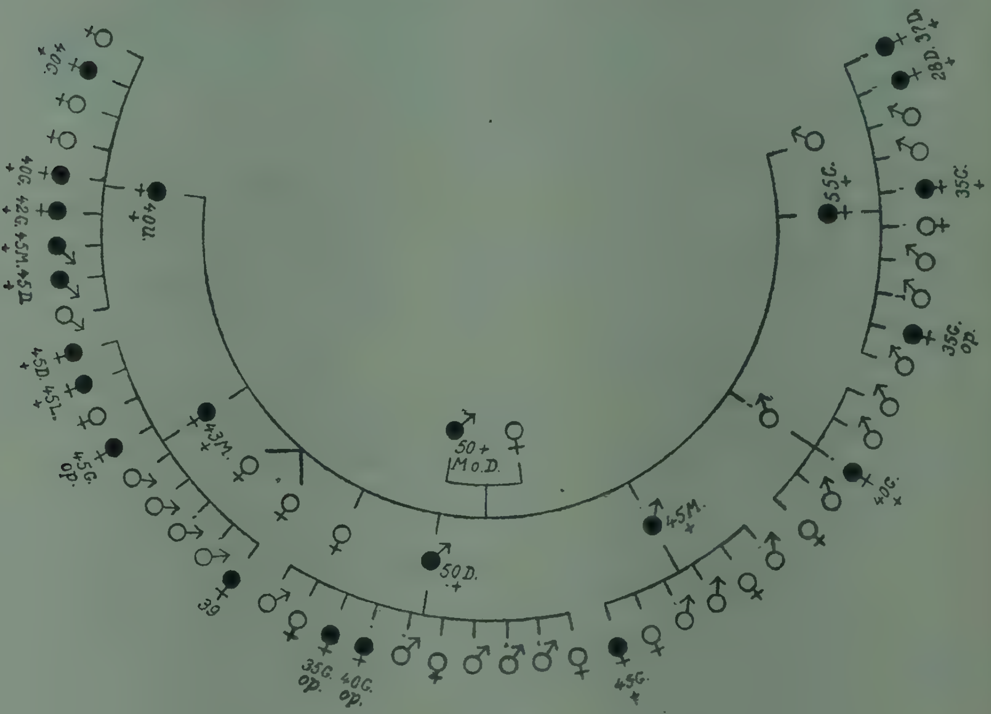

FIG. 131.

Cancer, after Warthin.

A. S. Warthin[1] has reported upon a very prolific family in which cancer was frequent. Here, among the children, grandchildren, and great-grandchildren of a man who died of cancer of the stomach or bowel, no less than twenty-seven have died of cancer. Inasmuch as most of the individuals belonging to the third generation of this family have not yet passed middle age, additional cases of cancer are to be expected. In Fig. 131 we see an extract from this genealogical tree. M. signifies stomach, D. intestines, G. uterus, L. liver, U. abdomen (exact seat unknown). In the members of this family the outbreak of the disease occurred on the average about fifteen years earlier than in members of the family described by Paulsen.

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[1] The Further Study of a Cancer Family, "Journal of Cancer Research", vol. IX, 1929, no. 2.

Cancer of the uterus is not so often found to run in families as cancer of the stomach. Nevertheless, in the family which came under Warthin's observation there had been no less than twelve instances of cancer of the uterus up to the date of record. Were gynecologists to make a closer study of the family history of their patients, it is probable that a family tendency towards cancer of the uterus would be more frequently detected.

Cancer of the breast, likewise, has often been observed to run in families. In Fig. 132, we see a genealogical tree showing the family incidence of cancer of the breast.[1] H. Leschcziner and others have also described such families.[2] According to W. F. Wassink, C. P. Wassink, and Van Raamsdonk,[3] who have studied the comparative frequency of cancer of different organs in women, among seven women suffering from cancer, approximately four suffer from cancer of the stomach, two from cancer of the uterus, and one from cancer of the breast. These authors found that in particular families where there was a tendency to cancer, there was also a tendency for the cancer to make its appearance in the members of each family in some particular one of the before-mentioned organs.

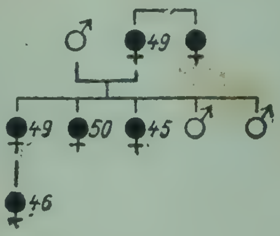

FIG. 132.
Cancer of the breast, after Wachtel.

Intestinal cancer, and especially *cancer of the rectum*, would seem also to run in families. In some of the cases the cancer develops as a sequel of hereditary polyposis intestinalis—a disease in which a varying number of innocent glandular tumours (adenomata) are formed in the gut, and in which one of these adenomata may ultimately become cancerous.

A tendency to the accumulation of cases in particular families has also been reported as regards lymphosarcoma, a malignant tumour originating in the cervical lymphatic glands. In this connexion I must refer once more to the leukaemias (see above, p. 370). These disorders are closely akin to the malignant tumours. The unrestricted and disastrous multiplication is the work of certain mother cells of the white blood corpuscles.

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[1] Communicated to me by H. Wachtel. See also this author's paper, Zur Frage der Erblichkeit des Krebses, "Münchener Medizinische Wochenschrift", 1924, no. 26.

[2] Ueber familiären Brustkrebs, "Medizinische Klinik", 1917, no. 21.

[3] Erblichkeit von Krebs, "Nederlandsche Tijdschrift vor Geneeskunde", vol. II, p. 326.

Markedly hereditary is *retinal glioma*, a rare malignant growth originating in the retina. About thirty families have been studied in which retinal glioma occurred in several brothers and sisters.[1] In a few of the instances, the trouble had also been observed in one of the parents. The observations hitherto made would lead us to believe, on the whole, that the heredity is recessive.

It is, however, a remarkable fact that in the genealogical trees hitherto published we note comparatively often that a majority of the children in any one affected generation have suffered from the disease: in one instance, ten out of sixteen (Newton); in another instance, eight out of eight (Wilson). It may of course be that family trees in which very many members of a family have been affected have been preferentially chosen for publication.

Additional light as to the nature of the hereditary predisposition to cancer may be devised from a study of the disease known as *xeroderma pigmentosum*, a recessive diathesis of the skin, which has already been considered on p. 271. The carriers of this diathesis ultimately develop, as a rule, cancer of the skin, the exciting cause being the influence of light. In xeroderma patients, sunlight, and to a lesser extent ordinary daylight, acts much as Röntgen rays act on the normal skin. We should note, also, that even in normal persons cancer of the skin rarely develops except in regions habitually exposed to sunlight and to chemical noxious influences.

When we take a general survey of the foregoing facts, we are, in my opinion, enabled to fit our knowledge concerning the origination of malignant tumours into a place in our general biological ideas, so that we can say that by its nature cancer is an idiokinesis (see pp. 465 et seq.) of somatic cells.

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[1] T. Leber, Ueber das Netzhautgliom, "Münchener Medizinische Wochenschrift", 1911, no. 49; W. Clausen, Vererbungslehre und Augenheilkunde, "Zentralblatt für die gesamte Ophthalmologie", vol. XIII, no. 4.

We are already acquainted with a great number of hereditary variations (mutations) which originate primarily in the germ cells. But if the idioplasm of the germ cells be subject to mutation, we can see no reason why mutation should not occasionally occur also in other cells of the body. In plants we are familiar with what are known as bud mutations, in which a cell undergoes a mutation of such a kind that it gives rise to a modified bud whose peculiarities are transmissible by inheritance. In like manner a human somatic cell which has been modified to become a cancer cell retains its peculiarities and transmits them by inheritance for countless generations during the growth of the cancerous tumour. In the study of various plants and animals we have become acquainted with certain races which exhibit an exceptional tendency towards mutations (see above, pp. 96–7). Manifestly, however, the immediate cause of mutations (idiokinesis) is external influences—such as sunlight in the case of xeroderma pigmentosum. Finally, where we have to do with influences which have a specially hurtful effect upon the nucleus (Röntgen rays, for instance), there is no need for the existence of a hereditary predisposition to malignant mutations, for here the external influences suffice unaided to produce the change. To that extent, therefore, as has been well said, every cell is "potentially cancerous".

There is considerable evidence to show that the abuse of alcohol may impair the hereditary equipment, and it seems probable that this poison is capable of injuring the idioplasm of somatic cells in such a manner that cancer is the outcome. According to Florschütz, among the persons insured by the Gotha Life Insurance Company during the fifty years from 1852 to 1902, there were 125 deaths from malignant tumours among publicans, and 121 deaths among brewers, per 100 deaths among the general run of the insured. A like noxious influence is exercised, presumably, by other idiokinetic poisons. We may well suppose that the increase in the prevalence of malignant tumours during recent decades may be partly accounted for by the increased working of such influences—(poisonous materials used in industrial operations, chemical adulterants added to foods, urban influences, treatment with

Röntgen rays). I write "partly" because we have to remember that some of the increase in cancer is due to the fact that in latter-day populations there is a larger proportion of elderly persons, and the elderly are peculiarly liable to cancer.

As regards the origination of cancer, I agree in general with T. Boveri,[1] who holds that the cause of malignant tumours is a disturbance of the normal chromosome constitution. He holds that such a disturbance usually depends upon the suppression of a cell division by noxious influences (pressure, poison). Owing to the suppression of the division, a diploid cell may become tetraploid; and when a tetraploid cell divides, we learn from experiment that abnormal chromosome combinations often arise.

Some people find it very puzzling that a cell which has hitherto been harmoniously adapted to the vital needs of the organism should suddenly become "malignant". I cannot myself see anything remarkable in this. From the early days of embryonic life, all the cells of the body are endowed with the faculty for growth and division. One might, rather, find it puzzling that when ontogenesis has been completed the cells should cease growing, but this cessation is obviously due to the working of some kind of regulative mechanism which comes into operation as a result of the attainment of a definitive stage of growth. Now, we have no reason to be surprised that these inhibitive mechanisms should sometimes be disturbed by external influences. Perthes,[2] the Tübingen surgeon, remarks aptly in this connexion: "Just as the movement of a watch is regulated by a particular organ, the balance, in such a way that an injury to this organ may make the watch move at an accelerated speed until it has run down, so in like manner an injury to particular parts of the cell nucleus may be the cause of the morbidly intensified frequency with which the division of the tumour cells occurs". Only in a comparatively small proportion of instances, of course, will a change in the idioplasm of somatic cells be of such a kind as to produce "malignant" growth. But other changes than this particular one will remain hidden, or will perhaps manifest themselves exclusively in the form of a weakness, a discoloration of the cells, etc. Those particular modifications, however, which result in an unrestricted power of multiplication will manifest themselves by the growth of malignant tumours.

We see, then, that the hereditary predisposition to malignant tumours may be of various kinds. First of all, it may depend upon a peculiar susceptibility of particular parts of the hereditary equipment to heredity-transforming influences—and this is what happens in cases of xeroderma pigmentosum. Secondly, particular morbific heredity factors may result in long-continued and repeated injuries to cells, until ultimately there ensues in the idioplasm of one of the cells a change leading to the growth of a cancer—and this is what happens in cases of hereditary gastric ulcer which undergoes transformation into cancer. Thirdly, however, we have to consider yet another possibility. From experiments on mice we learn that malignant tumours which can readily be transplanted among the members of a certain race, cannot be transplanted to the members of other and closely allied races.[1] We learn from this that there are heredity factors which can inhibit the growth of morbidly modified cells. It may be presumed, therefore, that the susceptibility to malignant tumours must partly depend upon a hereditary weakness of the power of resisting morbidly modified cells.

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[1] Zur Frage der Entstehung maligner Tumoren, Jena, 1914.

[2] Anent a discussion at the Medical and Natural Philosophical Society of Tübingen, see "Münchener Medizinische Wochenschrift", 1920, no. 3.

I trust that this conception of malignant tumours will not be regarded as a new discovery, but merely as a synthesis and unified formulation of already known facts. Only in relation to the dogma still occasionally voiced in various quarters that we do not "yet" know the "real cause" or the "essential nature" of cancer, may it signify a new standpoint (which, indeed, will not at first secure ready acceptance, seeing that it presupposes a familiarity with biological notions concerning heredity which is not as yet general among medical practitioners). The practical upshot of my view is the belief that cancer is of multiple causation. There is no longer any sense in attempting to discover a single specific cause for cancer in general. If we talk of "the" cause of cancer, we can only mean an element common to the various causes of cancer, and in this sense we may say that "the" cause of cancer is idiokinesis. By the same token, it is futile to search for a general cure of cancer. Still, we now have pointers which may guide us in the prevention of cancer: on the one hand, by the avoidance of idiokinetic injuries (for instance, Röntgen rays); and, on the other hand, by racial-hygienic measures.

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[1] Cf., for instance, C. C. Little, Factors influencing the Growth of a Transplantable Tumour in Mice, "Journal of Experimental Zoology" 1920, no. 3.

We must also bear in mind that Röntgen rays or radium emanation can not only give rise to cancer, but can also under favourable conditions cure the disease. We can explain these variable effects as follows. The nuclei that are especially influenced by the radiations are the nuclei of cells that are in course of division, whereas when a nucleus is quiescent the radiations will find considerably fewer points of attack. Inasmuch as, however, in rapidly growing cancerous tumours almost all the cell nuclei are perpetually dividing and redividing, whereas the nuclei of the other body cells are ordinarily at rest, it is possible to affect all the growing cancer cells by the radiations without seriously injuring thereby the normal cells whose nuclei are at rest. As a matter of principle, however, the production of the cancerous degeneration by the radiations and the destruction of cancer cells by the radiations are no more than two different degrees of one and the same process.

9. INCAPACITY FOR REPRODUCTION

"To talk about hereditary sterility is, properly speaking, to talk nonsense", wrote R. Hofstätter,[1] as late as the year 1926, and manifestly under the influence of a no longer tenable conception of heredity. The actual fact, of course, is that *sterility* or barrenness can very well be hereditarily determined, by recessive heredity factors. I expect that those cases of azoospermia (a lack of spermatozoids) which are not the outcome of such external noxious influences as gonorrhoea, are in the main hereditarily determined. Again, the male reproductive glands may be abnormally small or even altogether lacking, and this defect appears to arise upon an idiotypical foundation. The capacity for reproduction may also be impaired or destroyed by hypospadias (see pp. 297 et seq.). On the whole, however, in the male sex, heredity factors play a very unimportant part as compared with external noxious influences in the production of sterility.

According to P. Fürbringer,[2] Ashkenazic Jews are especially liable to be affected with "idiopathic" or "essential" sterility.

[1] Sterilität des Weibes, in Marcus' Handwörterbuch der Sexualwissenschaften, second edition, Bonn, 1926, p. 757.

[2] Sterilität des Mannes, Marcus' Handwörterbuch, etc., p. 747.

In such cases the testicles are to outward seeming normally developed, in especial the specific cells of the germinal epithelium. It is plain, moreover, that the sexual hormones are produced in normal fashion, but no spermatozoids are formed. We must presume recessive heredity factors to be the causes of this form of sterility.

In the female sex no less than in the male, gonorrhoea is the commonest cause of sterility, but morbific heredity factors are likewise of great importance. Defective development of the uterus has often been observed in several members of the same family. Above all, however, we find that *infantilism* (described above on p. 311 as a hereditary constitutional anomaly, and as usually having as one of its manifestations an under-development of the reproductive organs) leads to a marked impairment of the reproductive capacity. In a considerable proportion of sterile marriages, perhaps in Germany one third, the sterility is due to infantilism in the female partner. If there were no such disease as gonorrhoea, we can hardly doubt that more than three fourths of all cases of sterility would result from infantilism. Even when pregnancy occurs, as it sometimes does occur in the lesser degree of infantilism, we are apt to find that the uterus is unfitted for the proper development of the embryo, so that abortion takes place. Apart from the deliberate production of abortion, infantilism comes third on the list of causes of abortion, next to syphilis and morbid hereditary conditions of the embryo.

Long ago, F. Galton[1] observed a number of families among whose members the offspring were limited to one child apiece, and he regarded this comparative sterility as the outcome of hereditary predisposition. Seeing that, however, this "one-child sterility" is a typical outcome of gonorrhoeal infection in women, it is very difficult to ascertain whether, in any particular family, we have to do with hereditarily determined sterility or with an accidental impairment of fertility. Recently Lange has published two family trees showing the existence of this "one-child sterility" side by side with complete sterility.[2]

Hereditary are the growths known as *myomata*, non-malignant tumours which form in the wall of the uterus. Some of these may attain the size of a child's head, or more, and they may greatly impair fertility. The liability to the formation of these tumours runs in families, especially among Jews.

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[1] Hereditary Genius, London, 1869.

[2] Quoted by Agnes Bluhm, Zur Erblichkeit der Unfruchtbarkeit, "Archiv für Rassenbiologie", vol. XVIII, 1926, no. 4.

Of great importance to fertility in women is the formation of the pelvis, since during birth the child's head has to pass through this bony passage. In Germany, among all births, pelvic constriction causes difficult labour in from 3 to 5 % of the cases. Rickets is the commonest cause of constricted pelvis. We already know that rickets is in great measure hereditarily determined; but, apart from this, hereditary predisposition is in many cases directly responsible for the production of constricted pelvis. This remark applies especially to what is known as generally contracted pelvis.

There is certainly a direct connexion between the hereditary forms of the pelvis and other racial characters. The widest pelves are found in Nordics. Considerably narrower is the pelvis in women of Mongoloid race (we are especially well informed upon this point as regards Japanese and Malays). Smallest of all is the pelvis, on the average, in women of the Negroid races. Speaking generally, the shape and size of the maternal pelvis and the shape and size of the infantile head are mutually adapted. The obvious result of this will be that in mixed populations difficult labour from maladaptation in this respect will be peculiarly apt to arise. My own experience has taught me that when Swiss bovines, which are slender, wild-coloured animals, are crossed with stout black-and-white Dutch or East Frisian bovines, difficult labour is much commoner than in either of the parental races. No doubt the manifest luxurisation of the hybrids plays a part here. The same phenomenon can be observed in human hybrids, and may be a contributory cause of difficult labour in mixed human populations.

C. J. Gauss,[1] in observations made at the Freiburg Gynecological Clinic, found that a disproportionally large number of the women with contracted pelves came from the higher parts of the Black Forest, where a much larger percentage of the population is of the Alpine race (or, at any rate, not of the Nordic race) than in the low-lying regions adjoining the Rhine.

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[1] Ueber die Bedeutung der geographischen und sozialen Faktoren für die Aetiologie des engen Beckens, "Sitzungsbericht der Mittelrhein. Gesellsch. f. Geburtshilfe u. Gynäkologie", Frankfurt, 1912.

In the coastal areas of the North Sea and the Baltic, where the population is so predominantly Nordic, difficult labour due to contracted pelvis is much rarer. The greater frequency of contracted pelvis among the lower strata of the population may in part, at least, be due to similar racial differences. According to Agnes Bluhm,[1] in Baden during the year 1904 obstetric operations were required in 6·4 % of all births, whereas in Norway such operations are required in only 2·8 % of all births, although Norway is better supplied with obstetricians than Baden.

The *capacity to suckle* her offspring is of great importance as a part of a woman's competence for motherhood. If we follow Agnes Bluhm in accounting as not fully competent to suckle, all the women who are unable to give suck to their offspring for as long as six months after delivery, we find that about one third of German women are inadequately competent in this respect. Nor is complete incapacity to give suck by any means rare. Environmental influences are of little moment as factors of partial impairment or complete suppression of the function of lactation; and since, on the other hand, we find a marked tendency for such deficiencies to run in families, we can confidently infer that for the most part they are hereditarily determined. There is no trustworthy evidence in support of Bunge's view that incapacity for lactation is chiefly caused by parental alcoholism.

10. HEREDITARY DISEASES OF THE NERVOUS SYSTEM

When I speak of diseases of the nervous system, I refer especially to those diseases which produce bodily symptoms. The disturbances of the central nervous system which manifest themselves chiefly as mental anomalies will be described among the hereditary psychopathies, although by a euphemism it is customary to speak of these disorders and also of neurasthenia and hysteria as "diseases of the nervous system". A number of true nervous diseases have already been discussed, such as ptosis, optic atrophy, and glioma of the retina among eye troubles; auditory nerve atrophy among diseases of the ear; neuro-fibromatosis, Raynaud's disease, and hereditary chronic oedema, among diseases of the skin; Quincke's oedema, tetany, and migraine, among the diatheses. Epilepsy will be considered among the psychoses.

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[1] Zur Frage nach der generativen Tüchtigkeit der deutschen Frauen, "Archiv für Rassen- und Gesellschaftsbiologie", 1912.

Hereditary muscular atrophy is usually reckoned among diseases of the nervous system, although some physicians regard it as questionable whether muscular atrophy is primarily a nervous disease.

Most often, hereditary muscular atrophy appears as progressive muscular atrophy. Certain groups of muscles gradually become weaker and weaker, and while this increasing paralysis is setting in the affected muscles may actually grow larger owing to a simultaneous proliferation of the fatty tissue. At length the patient becomes completely helpless, and dies of the disease after years or decades. There are various kinds of muscular atrophy, distinguishable by differences in their heredity. In many families, the trouble begins in early childhood; in others, only in youth; in others, again, much later in life. In different families, moreover, the atrophy will tend to begin in different muscular groups: in one family, it may be, in the muscles of the pelvic girdle; in another, in those of the shoulder girdle; and in another, in those of the face. Some of these different forms are obviously transitional, and it has therefore been supposed by certain authorities that we merely have to do with varying outcomes of one and the same heredity factor. Strongly adverse to such a contention, however, is the fact that in the members of the same family the malady tends to run the same course—whereas (and this is still more

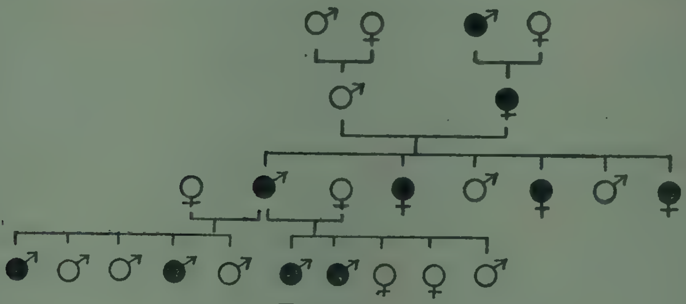

FIG. 133.
Progressive muscular atrophy, after Kehrer.[1]

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[1] F. A. Kehrer, Beitrag zur Lehre von den hereditären Muskelatrophien, a dissertation, Freiburg-im-Breisgau, 1908.

significant) it tends to run a very different course in persons belonging to different families. Comparatively frequent are dominant forms, and these take as a rule a relatively mild course; for in the case of dominant heredity, severe and rapidly progressing forms would not be able to maintain themselves in existence. In Kehrer's patients (see Fig. 133), in whom the heredity was dominant, the malady was exceptionally mild.

In Kehrer's tree the dominance of the heredity is made particularly striking by the fact that an affected man marries successively two healthy women and procreates with each of them both healthy and affected children.

Commoner are recessive forms, as shown in the results of a collective investigation inaugurated by Professor W. Weitz[1] in Tübingen. To the same category, we may suppose, belong likewise many of those cases in which isolated instances of progressive muscular atrophy have been observed in a family. Among 71 families in Weitz's investigation, 54 had "isolated" cases, 12 had "family groups" of cases, and only 1 exhibited "direct heredity".

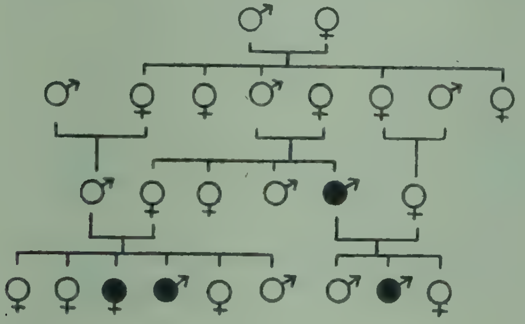

FIG. 134.
Progressive muscular atrophy, after Weitz.

If we assume that in each family on the average three children survive to an age at which the incidence of the disease becomes possible, then if the heredity were recessive we should expect according to the law of probability that the ratio would be 54 isolated cases to 20 + 4 family groups of cases. Weitz is inclined to ascribe the unduly frequent occurrence of isolated cases to the frequent appearance of appropriate mutations; for, inasmuch as most of the patients do not achieve reproduction, dominant mutants must usually remain isolated. For my part, I cannot regard it as likely that such mutations would be so frequent. Entres, in a research lasting for years, was unable to find any isolated cases of hereditary chorea, a typically dominant disease. If, therefore, isolated cases of a hereditary disease are frequently encountered, there is strong reason to believe that the heredity cannot be exclusively dominant. It is, of course, possible that some of the isolated instances were not inherited, but were the outcome of mutations in previously untainted stocks.

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[1] Ueber die Vererbung bei Muskeldystrophie, "Deutsche Zeitschrift für Nervenheilkunde", 1921.

Recessive sex-linked hereditary tendencies to muscular atrophy would also seem to be by no means rare. More commonly, however, these tendencies are simply recessive; and least often of all are they dominant.

Rarer than the progressive muscular atrophy with which we have hitherto been concerned is the disease known as *neural progressive muscular atrophy*, wherein the functional failure of the nerves is made manifest also in the form of recognisable changes in the affected nerves. In these cases, the muscular atrophy usually begins in those muscles of the foot which are supplied by the musculocutaneous nerve. Subsequently the muscles of the hands, the arms, and the legs become affected. In the general run of the cases, there are many more men than women. In some instances, at least, the heredity is recessively sex-linked.

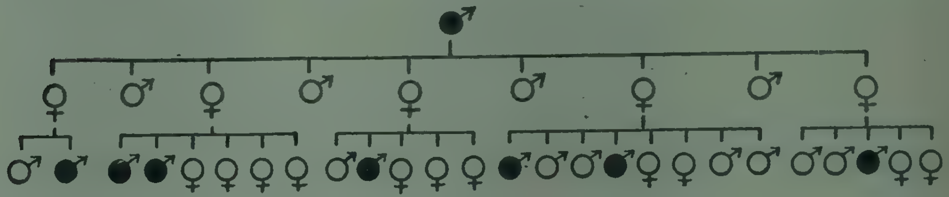

FIG. 135.
Neural progressive muscular atrophy, after Herringham.[1]

Akin to neural muscular atrophy is *spinal muscular atrophy*, characterised by wasting of the ganglion cells in the anterior horns of the spinal cord, with resulting atrophy of the axiscylinder processes of these cells. Spinal muscular atrophy usually begins in the fourth decade of life, the first muscles to waste being those of the hands. In this form of muscular atrophy we are apt to find that only one member of a family becomes affected, but often enough it has been observed in brothers, or sisters, or brothers and sisters. These facts indicate that the heredity is recessive. Unquestionably recessive is the variety of spinal muscular atrophy which begins in infancy and is occasionally observed in several children of a family.

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[1] Muscular Atrophy of the Peroneal Type affecting many Members of a Family, "Brain", vol. XI, 1889, p. 230.

To the same group belongs what is known as *bulbar paralysis*, wherein there occurs atrophy of the muscles of the tongue, the lips, the pharynx, the larynx, and the oesophagus—the immediate cause being atrophy of the ganglion cells of the medulla oblongata, by which the affected muscles are supplied. The patient loses the power of speaking and swallowing, usually dying within a very few years. Cases of bulbar paralysis are most often isolated, but sometimes we meet with two or three cases in brothers aand sisters. We may therefore presume that, like the other progressive muscular atrophies, the disease is hereditarily determined.

These progressive muscular atrophies belong to a group of disorders which result from a gradual atrophy of cells and tracts in the central nervous system. The changes in the peripheral nerves and in the muscles are sequels of this central change. We may refer the process to a premature senility of the affected parts, comparable to the premature greying of the hair and to certain other disorders which depend upon premature senescence of certain organs (diabetes, clouding of the lens, contracted kidney, etc.).

The disease known as *hereditary spastic spinal paralysis* results from atrophy of the pyramidal tracts (the columns of nerve fibres in the lateral regions of the white matter of the spinal cord). This illness usually begins in early life, taking the form of a peculiar stiff (spastic) paralysis of the legs, whereby unaided walking is made impossible. F. W. Bremer[1] has described a family in which spastic spinal paralysis could be traced in unbroken succession through six generations. Other authors, likewise, have described several instances in which the heredity of the disease was manifestly dominant. There seems, however, to be also a recessive taint which can manifest itself as spastic spinal paralysis. Recessive, certainly, was the heredity in a family described by Erb.[1] Pfaundler has reported upon a family in which all the six children of parents who were healthy, but closely related, developed spastic spinal paralysis in infancy. In the group of the hereditary spastic spinal paralyses we find, as so often, that the dominant forms are usually less severe, the recessive forms usually more severe (cf. Bremer).

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[1] Klinischer und erbbiologischer Beitrag zur Lehre von den Heredodegenerationen des Nervensystems, "Archiv für Psychiatrie", vol. LXVI, 1922, nos. 3 and 4.

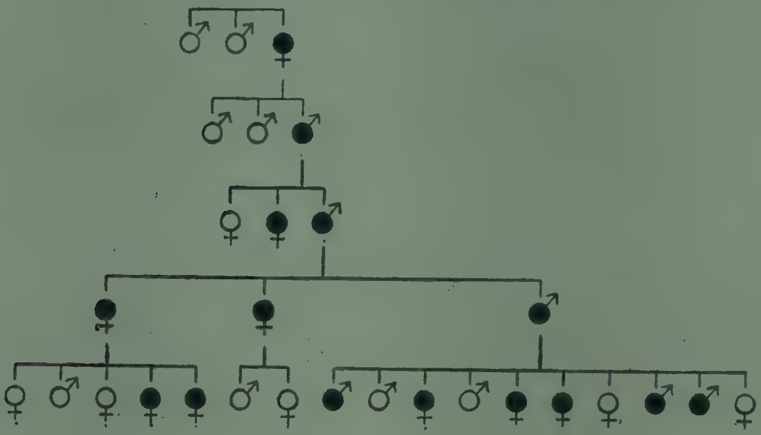

FIG. 136.
Spastic spinal paralysis, after Bremer (extract).

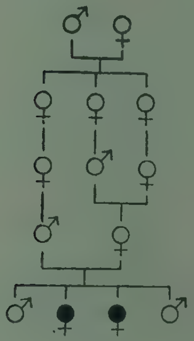

FIG. 137.
Spastic spinal paralysis, after Erb.

Some contend that spastic spinal paralysis may also be induced by external causes. We must, however, remember that an isolated case can very well be hereditarily determined. This fact has often been forgotten.

In what is known as *amyotrophic lateral sclerosis*, which resembles spastic spinal paralysis, there is atrophy of some of the ganglion cells of the anterior horns of the spinal cord as well as atrophy in the lateral tracts, with the result that muscular atrophy is associated with the spastic paralysis. The disease might, therefore, be more appropriately called spastic muscular atrophy. It has often been observed to run in families, and, on the other hand, there is no evidence that external causes play a part in its production.

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[1] "Deutsche Zeitschr. für Nervenheilkunde", vol. VI, p. 137 (quoted by Bremer).

As regards the morbid conditions classed together as "cerebral paralysis of children", caused by focal affections in the brain, and manifesting themselves by a peculiar stiffness of the legs, there can be no doubt that they also are hereditarily determined. When, as sometimes happens, the disease is already present at birth, it is usually termed "Little's disease" (spasmodic paraplegia of infants), and this would appear in most instances to be caused by injury to the brain during birth.

Beyond question hereditary is the progressive form of *spastic paraplegia* (paralysis of both sides, sometimes termed diplegia) which does not begin until after birth. This disease, which is characterised by stiffness of the legs, nystagmus, squint, decline in visual power, and failure of intelligence, seems to be recessive. Several forms have been described. Furthermore, the trouble tends to begin at different ages in different families—in infancy, in childhood, or even during the later years of youth. Fig. 138 reproduces a relevant genealogical tree.[1]

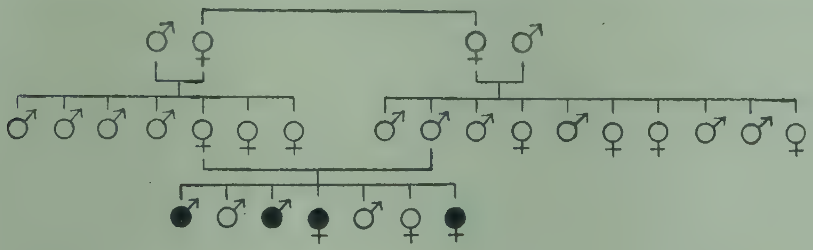

FIG. 138.
Spastic paraplegia, after Davidenko.

Should this illness happen to begin before birth, it would subsequently, we may presume, be classed as Little's disease, and we may therefore suppose that some, at any rate, of the cases classed under the latter head are hereditarily determined.

To the group of hereditary spastic paralyses belongs, also, a hereditary disorder first described by Pelizaeus and subsequently studied more exhaustively by L. Merzbacher.[2] So far it has only been observed in one kinship. The malady appears in early youth, beginning with tremor and disorders of movement, with which spastic paralyses and intellectual weakness are associated. The brain, it will be remembered, is made up of "grey matter" and "white matter", the grey matter consisting almost exclusively of ganglion cells, and the white matter almost exclusively of conducting fibres. Now, in Pelizaeus and Merzbacher's disease, owing to a lack of what are called the medullary sheaths of the nerve fibres, the white matter of the brain is imperfectly developed. The malady becomes manifest soon after birth, and usually proves fatal towards the end of the second decade of life. Although it is so rare as to be of little practical importance, it is of considerable theoretical interest, inasmuch as its sex-linked heredity proves that certain heredity factors contained in the sex chromosome contribute to the development of the brain.

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[1] Sent by S. Weissenberg to the "Archiv für Rassenbiologie", nineteenth year of issue, 1927.

[2] Gesetzmässigkeiten in der Vererbung und Verbreitung verschiedener hereditär-familiärer Erkrankungen, "Archiv für Rassen- und Gesellschaftsbiologie", 1909, no. 2.

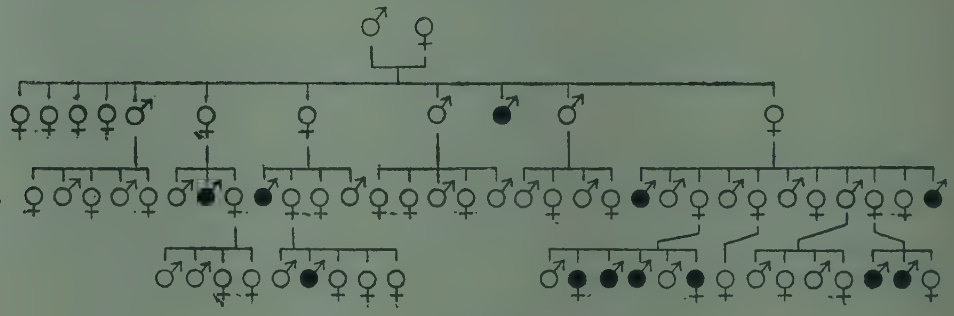

FIG. 139.
Pelizaeus and Merzbacher's disease.

A remarkable feature of this genealogical tree is that in the last generation two of the girls are among those affected. Since the father came of a healthy stock, these two persons cannot suffer homozygotically. We have to suppose, therefore, that the taint is not completely recessive, but may occasionally manifest itself heterozygotically also in females, just as has been observed in exceptional instances in the case of certain other recessive sex-linked taints.

In the case of *multiple scleroses* (in which focal disorders in the brain and spinal cord lead to progressive spastic paralysis and ultimately to dementia) a tendency to run in families has often been noticed, but scarcely more often than might in any case have been expected in instances of a disease which is by no means rare. The cause of the malady remains obscure. Kuhn and Steiner believe it to be an infectious disease caused by a spirochaete.

Beyond question hereditary, on the other hand, is the disease described by Wilson under the name of *pseudosclerosis*, which (ostensibly like multiple sclerosis) arises as the outcome of focal disorders of the brain, and is associated with cirrhosis of the liver (see above, p. 368). The malady appears to be recessive.[1]

We know very little as yet regarding the heredity of other kinds of "pseudosclerosis", such as the one described by Westphal and Strümpell, and the ones described by Jakob and Creutzfeld. The same remark applies to "*diffuse cerebral sclerosis*". A family tree published by Scholz[2] suggests that the heredity of diffuse sclerosis is recessive. A very remarkable point is that in this instance two of the grandfathers (they were brothers) of the affected children suffered from spastic spinal paralysis, but it seems doubtful whether there can have been any hereditary connexion between the illness of the grandparents and that of the grandchildren.

What is known as *tuberous sclerosis*, in which tuberous malformations arise in the brain, on the skin, and in various other organs, appears according to Siemens to depend upon a dominant taint which may manifest itself clinically in various ways (see above, p. 271).

The nervous disorders hitherto described are mainly the outcome of disease of the efferent nerve tracts, those which lead from the brain to the muscles; but there are also hereditary nervous disorders due to disease of the afferent nerve tracts, those leading towards the brain. To this category belongs, above all, hereditary spinal ataxia or *Friedreich's disease*. The trouble begins in the first or the second decade of life with disturbances of movement. The patient sways as he stands, and if he closes his eyes he is unable to stand at all, for the degeneration of the afferent nerve tracts has cut off the subconscious impressions which are among the essential factors upon which the maintenance of equilibrium depends.

E. Hanhart has done exceptionally good service in the study of the heredity of Friedreich's disease.[3] In Fig. 140 I reproduce one of his family trees.

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[1] Bielschowsky, Die Wilsonsche Krankheit, "Jahreskurse für ärztliche Fortbildung", May 1923, J. F. Lehmann, Munich.

[2] Klinische, pathologisch-anatomische und erb-biologische Untersuchungen bei familiäre diffuser Hirnsklerose im Kindesalter, "Zeitschr. f. d. gesamte Neurologie und Psychiatrie", vol. XCIX, 1925, no. 5.

[3] Beiträge zur Konstitutions- und Vererbungsforschung an Hand von Studien über hereditäre Ataxien, "Schweizerische Medizinische Wochen-

The heredity appears to be recessive. In 21 sets of brothers and sisters, Hanhart found that among 118 brothers and sisters 29 were diseased, this being 25 ± 4 %. The great frequency of blood relationship among the parents speaks even more strongly in favour of a recessive heredity. In Hanhart's 46 cases, 8, that is to say 17 ± 5 %, were the offspring of marriages of first cousins; and 26, that is to say 56 ± 7 %, were the offspring of the marriages of near kin. A study of all the information available would lead us to assume that in every instance of hereditary ataxia the sufferer's parents must have had a common ancestor even though it was several generations back, and that they must have received the recessive taint from this ancestor. We must therefore regard the malady as hereditary even when it affects only one member of a family. Consider, for instance, the family tree shown in Fig. 141, hitherto unpublished, and personally communicated to me by Hanhart. Here the ancestral pair from whom the recessive taint must have been handed down to both parents of the affected child, lived five generations back. Although

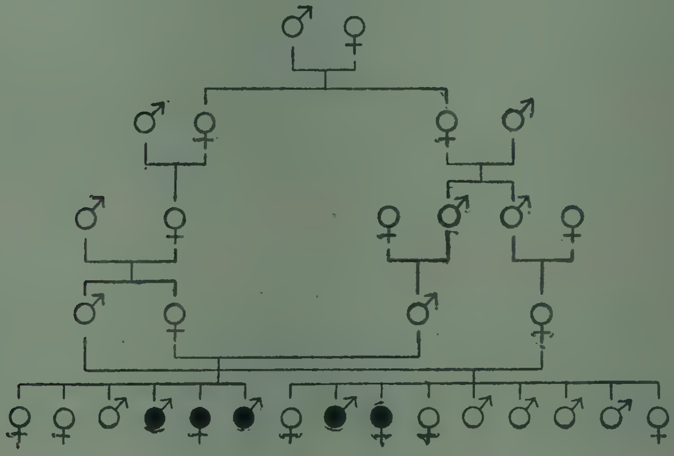

FIG. 140.

Friedreich's disease, after Hanhart (extract, modified).

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schrift", 1923, no. 6; Weitere Ergebnisse einer Sammelforschung über die Friedreichsche Krankheit in der Schweiz. "Schweizer. Archiv für Neurologie und Psychiatrie", vol. XIII, 1923, p. 297; Ueber die Bedeutung der Erforschung von Inzuchtsgebieten, "Schweizerische Medizinische Wochenschrift", 1924, no. 50.

the malady did not disclose itself either in these progenitors or in the intervening four generations, we must suppose that the taint was present in the kinship throughout the intervening period—and we cannot assume with any confidence that it originated in these progenitors in the middle of the eighteenth century, seeing that it may have been transmitted for hundreds of years before this without ever having become manifest. Indeed, we may consider it by no means improbable that all the cases of Friedreich's disease in Switzerland derive ultimately from a common source. Such a view is confirmed by the fact that the malady is not found everywhere, and that it is comparatively common in Switzerland. In the case of eighteen Swiss families in which Friedreich's disease occurs, Hanhart was able to trace back their origin substantially to four foci. He found an accumulation of the patients in certain remote valleys where inbreeding was common. "About half a dozen cases of Friedreich's disease are to be found at the present time in each one of a number of villages having a population of no more than a few hundreds. These patients are in the literal meaning of the term a heavy burden upon their relatives, for, in the advanced stage of this eminently chronic malady, they have completely lost the power of independent movement, and have to be carried about like helpless bundles."

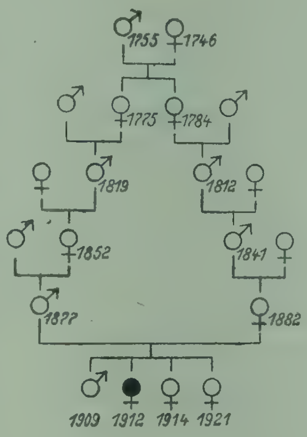

FIG. 141.

Friedreich's disease, after Hanhart. The figures in the illustration indicate the year of birth.

Over and above this severe recessive hereditary ataxia, there is to be observed in a few kinships a less severe hereditary ataxia having a dominant heredity. Such dominant ataxias usually exhibit the clinical picture of cerebellar paralysis (*cerebellar ataxia*) or Marie's disease, which depends upon atrophy of cerebellar tracts and manifests itself especially by a reeling gait. In most cases it begins during the third decade of life and advances very slowly. In the members of a family described by K. Classen,[1] the malady did not begin until

[1] Vererbung von Krankheiten und Krankheitsanlagen durch mehrere Generationen, "Archiv für Rassenbiologie", vol. XIII, 1918, no. 1.

the sixth decade of life, and here the heredity was plainly dominant. Sanger Brown[1] was able to trace the heredity of cerebellar ataxia through five generations of one family; there were 21 cases of the disease. W. R. Gowers,[2] too, has observed a dominant heredity in several families; and in England it would seem that, in general, dominant ataxia is commoner than recessive. In Sweden, E. Bergman was able to trace cerebellar ataxia through four generations of one family.[3] It would appear that of the dominant cases, too, some belonged to the spinal form. H. Triebel[4] has had under observation a family in whose members there occurred ataxia with a dominant heredity, some of the patients suffering simultaneously from spinal and cerebellar symptoms. Hanhart, moreover, among the Swiss families whose members suffered from Friedreich's disease, had one case in which the symptoms were predominantly cerebellar. It would seem, then, that the taint is modifiable, or that cerebellar hereditary ataxia and spinal hereditary ataxia overlap to some extent. But in all cases alike the rule applies that the severer forms are recessive and the slighter forms dominant in their heredity.

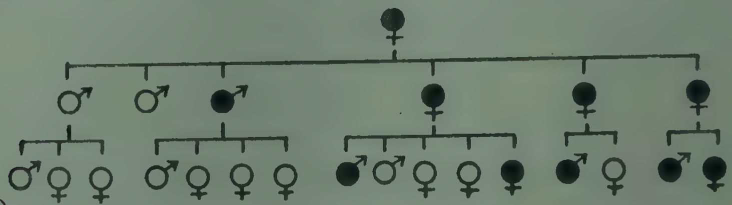

FIG. 142.
Cerebellar ataxia, after Classen.

In certain families affected by an apparently dominant ataxia, optic atrophy has been regularly observed to ensue in the course of the malady.[5] Apparently we are concerned here with a peculiar form of hereditary ataxia. The progressive atrophy of the afferent tracts of the spinal cord which is often a sequel of syphilis (tabes dorsalis or locomotor ataxia), occurring in adults, not unfrequently leads to optic atrophy—the optic nerves also consisting of afferent fibres.

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[1] On Hereditary Ataxy with a Series of 21 Cases, "Brain", vol. XV, 1892, p. 250.

[2] Diseases of the Nervous System, 1892.

[3] Studies in Heredo-Ataxia, "Upsala Läkareförenings förhandlingar", new series, vol. XXVI, nos. 5 and 6.

[4] Die Familie K., eine Studie über die Vererbung der Friedreichschen Krankheit, "Deutsche Zeitschrift für Nervenheilkunde", vol. LXXV, 1922, nos. 1 to 3.

[5] Cf. Wilbrand and Saenger, Handbuch der Neurologie des Auges, Erkrankungen des Optikus-Stammes, vol. V, Bergmann, Wiesbaden, 1913.

Paralysis agitans, also known as Parkinson's disease, is a not very rare malady which usually begins in the fifth or sixth decade of life, and manifests itself by the occurrence of peculiar involuntary movements which only cease during sleep. The hands, especially, are continuously moving as if hunting coins. There also develops a peculiar stiffness of the muscles, giving the face a mask-like look, and compelling the patient to assume an attitude bent a little forward. H. Lundborg[1] found in six out of seven patients that the parents had been consanguineous —but it must be noted that this was in a population where inbreeding was common. Whereas Lundborg's data favour the view that the heredity is recessive, we must not fail to note that there have been other observations pointing rather to a dominant heredity. Benedek and Csörsz[2] have reported on a family in which during four generations there were five unmistakable cases of the disease, three presumable cases, and three dubious cases. H. Kückens[3] has observed four cases in three generations of one family. Since twelve hitherto healthy members of the third generation have not yet passed the age at which the onset of the disease is common, it seems not unlikely that further cases will occur. Even if the heredity be dominant, we should in view of the late onset of the disease expect to find frequent interruptions in the heredity, seeing that many persons affected with the taint will certainly die before reaching the age at which paralysis agitans can manifest itself. Consequently, the nature of the heredity has not yet been clearly established. This much only is certain, that Parkinson's disease is mainly due to hereditary predisposition, and that environmental influences have little if any effect whether in the way of favouring or in that of hindering its onset.

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[1] Medizinisch-biologische Familienforschungen innerhalb eines 2232 köpfigen Bauerngeschlechts in Schweden, Jena, 1913.

[2] Heredofamiliarität bei Paralysis agitans, "Deutsche Zeitschrift für Nervenheilkunde", vol. LXXIX, 1923, no. 6.

[3] Ueber Heredofamiliarität bei Paralysis agitans, "Klinische Wochenschrift", 1925, no. 48.

According to M. J. Gutmann,[1] paralysis agitans is several times commoner in the Jewish than in the Gentile population.

Of a very different character are the involuntary movements in *Huntington's disease*, a hereditary form of chorea (St. Vitus's dance). The trouble begins in the fourth or fifth decade of life with twitching and often peculiarly stagey movements of the affected parts. The disease is incurable and a progressive dementia ensues. The heredity is simply dominant. Davenport[2] has reported upon 962 cases belonging to four kinships. The clinical features differed a little in the different families. In one of the families, there was scarcely any increase in the extent of the movements as time went on; in another, the malady began at an unusually early age; in a third, there was no accompanying dementia; in the fourth, on the other hand, mental weakness developed in the absence of choreiform twitchings. There would seem, therefore, to be several different morbific heredity factors at work, but all of them dominant. The investigations of L. Entres,[3] who has made an accurate record of the genealogy of all the families in Bavaria subject to Huntington's chorea, confirm the statement that the heredity is dominant.

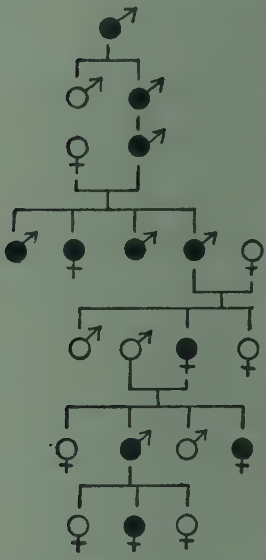

FIG. 143.
Huntington's chorea, after Entres.

Some authorities have declared that anticipation is characteristic of the heredity of Huntington's chorea, but Entres did not observe anything of the kind.

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[1] Die Rasse- und Krankheitsfrage der Juden, Müller and Steinicke, Munich, 1920.

[2] C. B. Davenport and E. B. Muncey, Huntington's Chorea in relation to Heredity and Eugenics, "Bulletins of the Eugenics Record Office", 1916, no. 17.

[3] Zur Klinik und Vererbung der Huntingtonschen Chorea, Springer, Berlin, 1921.

A transient *chorea* is not very rare as a sequel of infectious diseases, and especially of acute articular rheumatism. We have some reason to suppose that hereditary predisposition plays a part here, but have as yet no certain information concerning this matter.

Beyond question hereditary, however, is *chronic tic*, which, in contradistinction to Huntington's chorea, manifests itself already in youth in the form of twitching movements. It would seem, nevertheless, that the heredity of this disorder is not dominant.

In the same connexion I may refer to *progressive athetosis*, in which there occur choreiform movements, chiefly of the fingers and toes.

The name of *torsion neurosis* is given to a disease in which convulsive (clonic) backward movements of the head are observed. A genealogical tree published by Davidenko suggests a recessive heredity.

Heredity would appear to be a contributory factor in the production of *writer's cramp* and other "intention" or occupational spasms. In like manner, certain painful forms of cramp, such as cramp in the calves of the legs, have been observed to run in families.

Hereditary *tremor* occurs as a dominant anomaly. Hereditary tremor of the head associated with nystagmus has already been referred to in connexion with diseases of the eye.

Hereditary myotonia or *Thomsen's disease* is a malady in which all the muscles that are put in action after prolonged repose pass into a condition of extreme stiffness, and only relax after a considerable time. The immediate cause is an anomaly of the vegetative nervous system. In a family tree studied by Thomsen, and subsequently by his grandnephew K. Nissen,[1] we see an unmistakably dominant heredity. Here the disease has been traced uninterruptedly through seven generations.

In a family observed by Stattmüller,[2] myotonia began at the age of puberty, but disappeared almost completely when the patients were a few years older. In this family, likewise, the heredity was dominant.

In *paramyotonia*, described by Eulenburg, the muscular stiffness only ensues after exposure to cold. This kind of myotonia, too, could be traced uninterruptedly in one family through six generations.

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[1] Beiträge zur Kenntnis der Thomsenschen Krankheit, "Zeitschrift für Klinische Medizin", vol. XCVII, 1923, nos. 1 to 3.

[2] Beobachtungen an einer Familie mit Thomsenscher Krankheit, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. LXXXI, 1923, nos. 1 and 2.

Myotonic dystrophy (also called myotonia atrophica or Steinert's disease) usually begins in the third or fourth decade of life with myotonic manifestations in the hands and with the appearance of a peculiar kind of cataract.[1] When the patient grasps an object, to pick it up or for some other purpose, a spasm ensues and he cannot release his grip. In association with spasmodic and atropic manifestations in various groups of muscles, there ultimately ensues a general decay, and the unhappy patient dies prematurely. Here symptoms of myotonia and myatrophy, and disturbances of the vegetative and the animal nervous system, are associated with disorders of internal secretions (atrophy of the reproductive glands). The primary seat of the mischief is probably in the central nervous system. Now that attention has been directed to myotonic dystrophy, within the last few years 200 cases have come under observation. Although several genealogical trees have been drawn up, the nature of the heredity has not yet been clearly ascertained.[2]

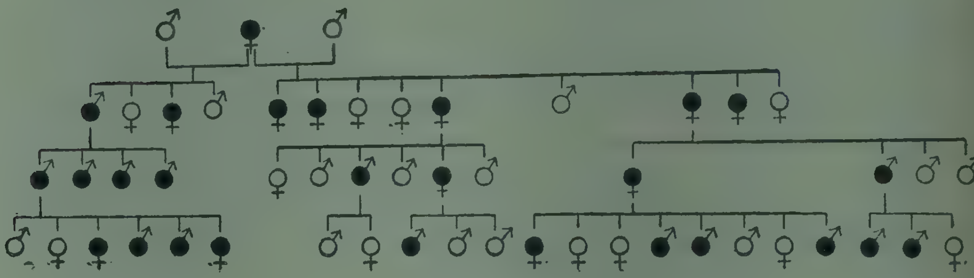

FIG. 144.

Myotonia (Thomsen's disease), after Nissen (extract).

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[1] A. Vogt, Die Cataract bei myotonischer Dystrophie, "Schweizerische Medizinische Wochenschrift", 1921, no. 29.

[2] B. Fleischer, Untersuchungen von sechs Generationen eines Geschlechtes auf das Vorkommen von myotonischer Dystrophie, "Archiv für Rassenbiologie", vol. XIV, 1922, no. 1; Zur Vererbung nervöser Degenerationen, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. LXXXIV, 1923; C. Frey, Beitrag zur myotonischen Dystrophie, "Archiv für Rassenbiologie", vol. XVII, 1925, no. 1.

It is reported that this morbid state, which now that the case records have been collected can be traced back for eight generations, has manifested itself more and more severely in successive generations. In the earliest generations there were no morbid symptoms of this particular kind. In the intermediate generations, the carriers of the taint suffered mainly from cataract. Only in the latest generations has the complete picture of myotonic dystrophy appeared. Furthermore, in the latest generations, the trouble has begun earlier in life and has been more severe than in previous generations. Thus there is said to have been both "anticipation" and "progression". Such a course of events would be the outcome of that "progressive degeneration" which Morel and some other physicians of an earlier day believed to occur; and to some extent it would correspond with what many palaeontologists have spoken of as "orthogenesis". For my part, however, I think we should do well to wait until further investigations have been made before assuming that obscure processes of this character must be invoked to account for the heredity of myotonic dystrophy. It seems to me probable that a far simpler explanation will be found. Inasmuch as myotonic dystrophy has only of late years been clearly distinguished as a pathological entity, and (though by no means rare) escaped the notice of physicians of earlier generations, it seems likely enough that there were cases of the malady classed under some such vague term as "exhaustion", "consumption", "cachexia", etc. If this be so, we may be concerned here with a mainly dominant taint which manifests itself more severely or less severely according to the way in which it is combined with other hereditary taints.

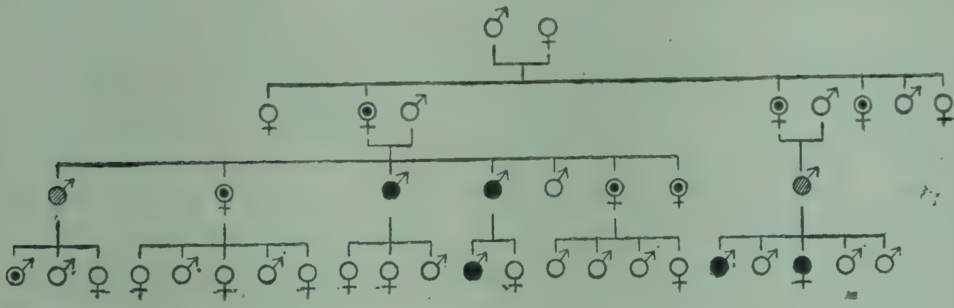

FIG. 145.

Myotonic dystrophy, after Frey (extract).

Hatching denotes a case of illness whose nature could not be clearly established, but which was perhaps myotonic dystrophy. A black dot within a circle signifies cataract.

The name of *trophoneurosis* has been given to a rare disease characterised by the formation of ulcers and atrophic areas in the skin of the feet, especially of the toes, and sometimes manifesting itself in the hands as well. This malady has as yet been seen only as a family trend (usually appearing in brothers and sisters), and we must therefore suppose the heredity to be recessive.[1]

In the group of trophoneurotic disorders we may also class *trophoedema*, *sclerodermia*, *erythromelalgia*, *Raynaud's disease*, and similar affections, whose heredity has in part been already discussed in connexion with diseases of the skin, and in part still remains obscure.

Related to the same group of disorders is *syringomyelia*, which obviously arises as the outcome of an inborn taint, and may therefore be supposed to be the expression of a particular hereditary predisposition. In this malady there are cavities in the spinal cord, especially in the cervical region, leading by slow degrees to progressive nervous disturbances, especially in the hands (anaesthesia, loss of temperature sensation, trophic disorders). F. W. Bremer[2] has made an exhaustive study of its family prevalence, and has been able to show that among the relatives of those suffering from marked forms of the disease, there are to be found less conspicuous forms and isolated symptoms of the malady. Following Henneberg, he believes that we can detect a peculiar constitutional type to which may be given the name of "status dysraphicus", and out of which (owing to the operation of causes which are still obscure) the definite clinical picture of syringomyelia may occasionally develop. The status dysraphicus seems to be often associated with crooking of the fingers, funnel-breast, and spinal curvature. In a pair of twin sisters, Weitz found only one of the girls suffering from syringomyelia. It is clear from this that environmental factors are needed for the production of the disease, in addition to hereditary predisposition.

In the well-known family tree published by S. Goldflam,[3] the cases of paroxysmal muscular paralysis (*paraoxysmal myoplegia*) following exposure to cold, exhibited a heredity that was irregularly dominant.

Transient paralysis of particular nerves (such as the facial nerve and the musculospiral nerve, Bell's paralysis, etc.) following minor infections such as a cold in the head, tend obviously to run in families. Kehrer, in the before-mentioned work, refers to family *hypertrophic neuritis* as a hereditary nervous disorder.

Hereditary predisposition appears to be an important factor of *muscular rheumatism*, a painful inflammatory condition of certain muscular nerves. The same remark applies to what is known as

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[1] W. Weitz, Kasuistisches zur familiären Trophoneurose an den Füssen und Händen, "Deutsche Zeitschrift für Nervenheilkunde", vol. LXXXII, 1924, nos. 1 and 2.

[2] Klinische Untersuchungen zur Aetiologie der Syringomyelie, "Deutsche Zeitschrift für Nervenheilkunde", vol. XCV, 1927, nos. 1 and 2.

[3] Weitere Mitteilung über paroxysmale familiäre Lähmung, "Deutsche Zeitschrift für Nervenheilkunde", vol. VII, 1895, nos. 1 and 2.

lumbago. Intermittent lameness (claudicatio intermittens), which in earlier days was ascribed to a peculiar localisation of arteriosclerosis, occurs almost exclusively among Jews. We may presume, therefore, that it is a hereditary nervous disorder. The abuse of tobacco would seem to be a contributory factor in its production. Alikhan[1] has observed a hereditary lack in the power of smell (*anosmia*) in several members of the same family. This anomaly, which apparently depends upon a congenital lack or a congenital functional incapacity of the olfactory nerves, is a parallel disorder to hereditary deaf-mutism and to hereditary absence of the optic nerves.

In connexion with anomalies of the nervous system allusion must be made to nocturnal incontinence of urine (*enuresis nocturna*), a very troublesome and distressing complaint. It tends to run in families, and is manifestly hereditary. Persons who suffer from it are comparatively often feebleminded or epileptic, and they are frequently affected with malformation of the spinal cord (spina bifida occulta). Jancke[2] was able to trace nocturnal incontinence of urine through four generations of a family; in three of the members of the family, spina bifida could be detected on X-ray examination.

The disturbances of speech included under the names of *stuttering* and *stammering* tend markedly to run in families. In the famous Juke kinship there is a stutterers' family tree which suggests a dominant heredity.[3] According to F. A. Bryant,[4] about one half of all stammerers have relatives who stammer. In the population of Germany, approximately 1 % of all persons stammer. It is a mistake to suppose that when the children of stammering parents stammer, the disorder is always the result of imitation, for cases have been observed in which the children of stammering parents were separated from their parents from the time of birth, but nevertheless became stammerers.

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[1] L'épilepsie et l'anosmie héréditaire, "Schweizerische Medizinische Wochenschrift", 1920, p. 211.

[2] Ueber eine Bettnässer familie; zugleich ein Beitrag zur Erblichkeit der Spina bifida, "Deutsche Zeitschrift für Naturheilkunde", 1915.

[3] A. H. Estabrook, The Jukes in 1915, Washington, 1916.

[4] Influence of Heredity in Stammering, "Journal of Heredity," vol. VIII, 1917, no. 2.

In stammerers and their relatives, psychopathic conditions of various kinds (epilepsy, hysteria, feeble-mindedness) are met with in above-average frequency. In mon-oval twins. stammering has several times been observed in both members of the pair; but in other mon-oval twins, Siemens has observed stammering in only one of a pair.

Among disorders of speech there exists, apart from deaf-mutism, a condition in which children which can hear perfectly well do not begin to speak at the usual age. This delay in the development of the faculty for speech tends to run in families. It is technically known as "*idiopathic dumbness*". In more than one third of the cases we find that one or other of the parents began to speak late, or exhibited some other defect in the development of the faculty of speech.

Heredity is likewise an important factor of *lisping*, a disorder of speech in which the point of the tongue is brought too close to the upper incisors when sibilants are being pronounced. H. Gutzmann[1] found that in "lateral lisping" (sigmatismus lateralis) the same defect of speech was present in 38·5 % of the kin of those suffering from the affection. Many Jews are inclined to have a slight and peculiar lisp. This may be a hereditary racial trend in certain elements of the Jewish stock, and in that case it can hardly be regarded as abnormal.

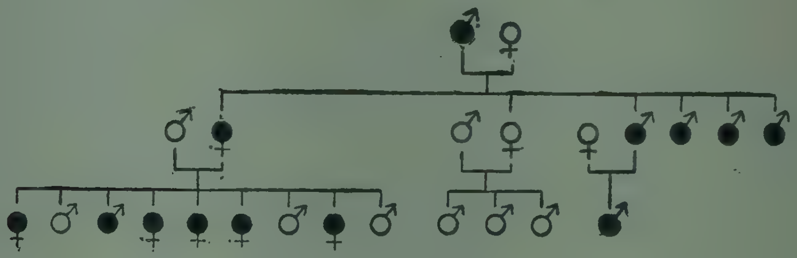

FIG. 146.

Stammering, after Estabrook (extract from the Juke family tree).

Hitherto observers have, in general, failed to recognise or to allow sufficiently for the fact that the various nervous diseases as clinically distinguishable do not in general represent unified bio-types, but are complexes of a number of hereditary types (cf., for instance, the muscular atrophies and the ataxias). The tendency has, rather, been to move altogether away from this idea of biologically unified hereditary types of nervous disorder, and to suppose that there is a kind of generalised "heredodegeneration", which manifests itself now as one clinical picture and now as another. This pompous term "heredodegeneration" is based upon the somewhat muddle-headed views of the neurologist E. Jendrassik,[1] and upon the following misinterpreted observations: first, that occasionally we encounter clinical pictures which seem to represent intermediate or transitional forms between other clinical types (for instance, myotonic dystrophy); secondly, that, within one family, differences in the clinical picture of a disease may present themselves (for instance, cerebellar ataxia in one member side by side with spinal ataxia in another); and, thirdly, that, within any one family, maladies belonging to entirely different groups have been observed (for instance, spastic spinal paralysis in one member side by side with diffuse sclerosis in another). But this doctrine of a unified "heredodegenerative" predisposition is adequately refuted by the simple fact that in numerous instances we have already succeeded in tracing the heredity of very sharply definable morbid taints. The doctrine of "heredodegeneration" in the domain of diseases of the nervous system is on all fours with the doctrine of the "status degenerativus" in the domain of anomalies in the habit of body (see above, p. 312); and the circumstances which have led to the formulation of the one hypothesis can be accounted for on the same lines as the circumstances which have led to the formulation of the other. Although a particular morbific hereditary taint may within certain limits manifest itself differently in different members of the same family (especially when it is combined with other and not necessarily morbific hereditary predispositions), it by no means follows from this that all hereditary nervous disorders are due to one and the same morbific heredity factor. Insofar as the coincidence of essentially distinct hereditary nervous disorders in one family is dependent upon anything more than chance, a genuine aggregation of such distinct troubles in one family may arise: first, from inbreeding (a manifestation of various recessive troubles as a result of consanguineous marriages); and, secondly, from social and sexual selection, inasmuch as persons with various kinds of hereditary defects are not likely to be chosen as mates by normal human beings, and therefore have to content themselves with one another.

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[1] Die Vererbung von Sprachstörungen, contributed to Noorden and Kaminer's Krankheiten und Ehe, Leipzig, 1916, p. 470.

In answer to the possible objection that the word "heredodegeneration" is not intended to signify a unified predisposition, but is nothing more than a comprehensive designation for the generality of hereditary disorders, I should like to say that such a lumping together would be no more expedient than is the aggregation of all diseases due to external influences under the term "exogenic disease" (Kehrer). In any case, the word "heredodegeneration", which has come into use in other specialties besides that of the neurologists, is an undesirable one. If the composite term "degeneration" signifies the appearance of hereditary factors of disease, then "heredodegeneration" is tautologous; and if, on the other hand, "degeneration" here means nothing more than the breakdown of organs from internal causes, thus signifying the same thing as the equally pretentious word "abiotrophy", the term leads us positively astray.

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[1] Die hereditären Krankheiten, contributed to Lewandowsky's Handbuch der Neurologie, Berlin, 1911, vol. II.

Unfortunately we still lack a systematic presentation of hereditary nervous disorders penned by a competent neurologist and at the same time inspired by a modern outlook. Jendrassik's account is biologically inadequate. F. Kehrer's attempt in this direction[1] does not claim to be and cannot be regarded as more than provisional.

11. HEREDITARY INSANITIES AND PSYCHOPATHIES

In no other group of diseases is heredity so conspicuous among the causes as it is in the case of mental disorders, but at the same time there is no other field in which the study of heredity is so difficult. For, to begin with, specialists are far from being agreed as yet upon the classification and delimitation of the hereditary diseases of the mind. We need hardly be surprised, therefore, that in scarcely one of these diseases has the hereditary course been satisfactorily established. Only this much is certain, that heredity is the outstanding cause of mental disorders.

Second among the chief factors comes syphilis, for it is not only responsible for the production of general paralysis of the insane, but also in its congenital form can induce feeble-mindedness and epilepsy. In the third place among the main causes of mental disease comes alcohol, producing delirium tremens and alcoholic insanity in persons actually given to drink, while in their children it may cause feeble-mindedness and other mental disorders.

For practical purposes we divide mental disorders into the *insanities* proper (*psychoses*) and the *psychopathies*, the classification being determined with reference to conduct, to the sufferer's retention of the capacity for adaptation to daily life or to his retention of the power of self-preservation. Speaking generally, we must regard as mentally abnormal or mentally disordered any individual whose capacity for adaptation is considerably reduced on account of his mental state. When the disturbance is so severe that the sufferer cannot follow an ordinary occupation, we speak of insanity. In slighter degrees of disturbance, when the sufferer is able to pursue some ordinary occupation, but is in a condition which either permanently or occasionally reduces his powers of self-preservation or his fitness to fulfil his obligations to society, we speak of psychopathy.

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[1] Die erblichen Nervenkrankheiten, "Deutsche Zeitschrift für Nervenheilkunde", vol. LXXXIII, 1924, p. 201.

This classification, which is in conformity with the demands of practical life, corresponds very little to the needs of the science of heredity. The aim of the geneticist must be to recognise the varying manifestations of the particular morbific heredity factors, and to determine the course of their heredity. We may hope that in due time genetics will enable us to classify mental disorders in accordance with their essential nature—a desideratum which clinical study has as yet imperfectly fulfilled. At the start it is obvious that there must be a number of different hereditary mental disorders. Mental processes are expressions of the functions of the brain, and the brain, like any other organ, is subject to numerous hereditary anomalies.

Inasmuch as a classification of mental disorders based upon genetics is not yet possible, the classification adopted in the ensuing pages (modified from the traditional one) cannot be regarded as definitive. Authorities for whose opinion I have great respect have pointed out to me that it is impossible to convey an intelligible picture of the various mental disorders to the laity within the compass of a few sentences. Nevertheless, I cannot write at great length upon this matter; and on the other hand I do not wish to content myself with the use of mere names that would carry no meaning to the laity. I therefore sketch my definitions in such a way as to remind medical practitioners who lack specialist training in these matters of the most important characteristics of the diseases in question, and in such a way as to give those without any medical training at all some vague idea of the severity and the characteristics of the various conditions. But I neither expect nor desire to enable the layman to recognise these diseases when he comes across instances of them in daily life.

In earlier subsections I have occasionally referred to mental disorders, for instance, as an accompaniment of hereditary chorea; but these conditions were classified among nervous disorders because the bodily rather than the mental symptoms stood in the foreground. Strictly speaking we have to do with mental disorders likewise in the case of deafness, colour-blindness and anosmia, for in all these there is a lack of certain sense impressions, whilst we know little or nothing about the physical side of such troubles. Like considerations apply to migraine and to other hereditary forms of headache. It follows that these troubles ought really to be included among disorders of the mind.

Several kinds of congenital feeble-mindedness are classed together under the names of *imbecility* and *idiocy*. When, notwithstanding a considerable degree of mental enfeeblement, an individual remains capable of earning his livelihood, he is no longer spoken of as an imbecile.[1] The imbecile, though incapable of earning an independent livelihood, can be trained to perform under supervision certain kinds of useful work, whereas an idiot is incapable of this degree of training. We see, then, that the distinction between an idiot and an imbecile is only one of degree. Obviously there will be transitional cases between idiocy and imbecility; and within the respective groups of idiots and imbeciles there will, again, be numerous gradations. Feeble-mindedness may be the expression of very varied morbid states, some of which are the outcome of inheritance, whilst others are caused by external influences. For this reason we are not entitled to speak without qualification of the heredity of imbecility or of idiocy.

A notable proportion of the cases of feeble-mindedness are due to congenital syphilis. According to Kraepelin, at least one third of the severer forms of idiocy are the outcome of syphilis. Parental alcoholism is perhaps an equally important cause of grave idiocy. In still greater measure, however, is feeble-mindedness a part manifestation of cretinism. But, after deducting all the cases which are due to external noxious influences, there is left a majority of cases of feeble-mindedness, and these cases are of an essentially idiotypical nature. The American investigator H. H. Goddard[2] has come to the conclusion that at least two thirds of all cases of feeble-mindedness are hereditarily determined. This estimate harmonises well enough with our own experience in Germany, when we remember that in the United States alcoholism does not play a very large part, and that there both syphilis and cretinism seem to be rarer than they are in Germany. In the

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[1] In the English-speaking world, and especially in the United States of America, such comparatively high-grade mental defectives are now usually termed "morons".—TRANSLATORS' NOTE.

[2] Feeble-mindedness, New York, 1914.

Kallikak family described by Goddard,[1] the offspring of 40 marriages in which both the parents were imbeciles numbered 222, and of these 220 were feeble-minded and 2 apparently normal. As the outcome of marriages between feeble-minded persons and normal persons, only about half of the offspring were feeble-minded. Feeble-mindedness could be traced in unbroken succession for five generations. This evidence shows that there must be dominant heredity factors of feeble-mindedness.

When C. B. Davenport and D. F. Weeks[2] come to the conclusion that feeble-mindedness is the outcome of a recessive taint which can simultaneously determine epilepsy, this is beyond question an unjustified generalisation. A comprehensive view of the facts shows that feebleness of mind is no more a biological unity than is feebleness of body. It is, of course, possible—nay probable—that some of the forms of feeble-mindedness depend upon a recessive taint; and since feeble-minded persons often intermarry (being usually unable to procure normal mates), it is natural that even when the taint is recessive the heredity of feeble-mindedness should frequently be traceable through several generations.

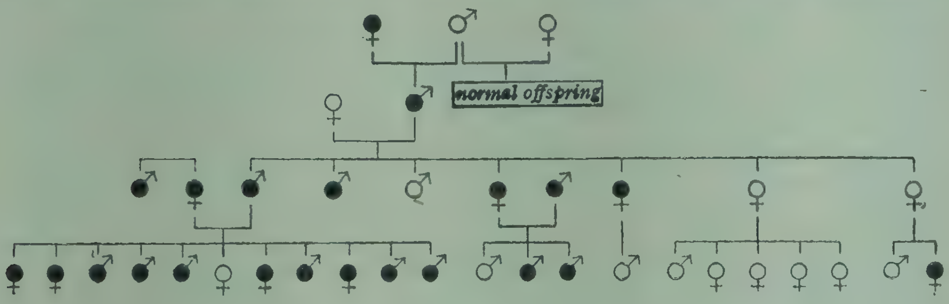

FIG. 147.
Imbecility, after Goddard (extract from the Kallikak family tree).

In another investigation, conducted jointly with F. H. Danielson, Davenport obtained much the same results as Goddard.[3] In the great kinship described as the "Hill Folk", when two feeble-minded persons mated, about three fourths of their children were feeble-minded, whereas if the heredity had been simply recessive we should have expected all the children to be feeble-minded. When one parent was normal and the other feeble-minded, about half of the children were feeble-minded; and when both parents were normal but were themselves the offspring of feeble-minded persons, about one third of their children were feeble-minded.

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[1] The Kallikak Family, New York, 1912.

[2] A First Study of Inheritance in Epilepsy, "Eugenics Record Office Bulletin", 1911, no. 4.

[3] F. H. Danielson and C. B. Davenport, The Hill Folk, "Eugenics Record Office Bulletin", no. 1, 1912.

We are indebted for an instructive study to the Rostock hygienist Reiter.[1] He and his collaborator Osthoff found that of 250 children at the Rostock school for feeble-minded children, 60 (24 %) had had feeble-minded fathers, 80 (32 %) had had feeble-minded mothers, and 29 (11·6 %) had had feeble-minded fathers and mothers. Thus at least 67·6 % had feeble-minded ancestry. I say "at least" since we cannot tell whether all the parents not positively known to have been feeble-minded were really equipped with normal intellectual powers. Of the 140 children one or other of whose parents had been feeble-minded, 103 (73·6 %) had one or more feeble-minded brothers or sisters. The 29 children both of whose parents were feeble-minded all had feeble-minded brothers or sisters; and among the brothers and sisters of the children in this group 90·7 % were feeble-minded and only 9·3 % normal. In two families where one of the parents was feeble-minded, there were 8 feeble-minded children. These data favour the view that there are dominant heredity factors of feeble-mindedness. But among 102 feeble-minded children who could not be shown to have had feeble-minded parents, 64 % had feeble-minded brothers or sisters, which suggests the cooperation of recessive heredity factors.

Frau Rüdin-Senger obtained similar results from a study of the children in the Munich school for the feeble-minded.[2]

The view that feeble-mindedness may also be caused by recessive heredity factors is supported by the fact that idiots are born in above-average proportions as the offspring of the marriages of near kin. According to the investigations of A. H. Huth,[3] idiotic, imbecile, of high-grade feeble-minded children were found in the following proportion among the offspring of the marriages of near kin:

Gralhaus	3·8 % of	1388	children
Howe	4·7 % ,,	359	,,
Down	7·0 % ,,	852	,,
Ireland	8·5 % ,,	213	,,
Connecticut Commission . .	12·5 % ,,	160	,,
Bemiss	15·0 % ,,	?	,,
Mitchell	18·1 % ,,	519	,,

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[1] H. Reiter and H. Osthoff, Die Bedeutung endogener und exogener Faktoren bei Kindern der Hilfsschule, "Zeitschrift für Hygiene", vol. XCIV, 1921.

[2] E. Rüdin, Ueber Vererbung geistiger Störungen, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. LXXXI, 1923, nos. 3 and 4.

[3] The Marriage of Near Kin, second edition, London, 1887.

Unfortunately since Huth's day there has been no systematic study of the offspring of the marriages of near kin.

It would seem, then, that there must be recessive as well as dominant heredity factors capable of producing feeble-mindedness; and in this instance, likewise, the rule applies that in the slighter degrees of the affection the heredity is usually dominant, and in the graver degrees it is usually recessive. Manifestly the heredity of idiocy cannot well be dominant, seeing that idiots so seldom are able to reproduce their kind.

This last consideration explains the results obtained by T. Ziehen,[1] who found in the case of children suffering from the slighter degrees of feeble-mindedness that a very large percentage of them had had feeble-minded parents, whereas in the case of children suffering from the graver forms of idiocy, evidence of parental taint was usually lacking. When the heredity is recessive, we may expect to find that the parents were healthy. We have also to remember that high-grade feeble-mindedness may be the outcome of congenital syphilis.

Since the normal differences of faculty are polymerically determined, we may infer that the slighter degrees of feeble-mindedness (those bordering on the normal) will likewise be to some extent polymeric; but this does not exclude the possibility that there may be, in addition, certain abnormal heredity factors of which a single one (at any rate when present homozygotically) may be competent to produce high-grade feeble-mindedness.

The mothers of feeble-minded children are more often found to have been feeble-minded than the fathers. This is manifest in Reiter and Osthoff's material; and it is plainer still in Schott's[2] material and in Schlesinger's.[3] Various causes may account for this difference. First of all, it is possible that the heredity factors of feeble-mindedness may display their working more clearly in women than in men; secondly, feeble-minded men less often succeed in achieving marriage than do feeble-minded women, since under present conditions higher demands in respect of intelligence are made upon men than upon women; thirdly, some of the heredity factors which determine feeble-mindedness may be sex-linked.[1]

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[1] Die Geisteskrankheiten des Kindesalters, Reuther and Reichard, Berlin, 1915.

[2] Ueber die Ursachen des Schwachsinns im jugendlichen Alter, "Archiv für Psychiatrie", 1919.

[3] Schwachbegabter Schulkinder, Stuttgart, 1907.

Noteworthy is the fact that congenital feeble-mindedness is apt to betray its presence in bodily characteristics as well. Every one knows what an idiot looks like; and though in the slighter degrees of feeble-mindedness the bodily characteristics may not be obvious in individual instances, even the layman will have no doubt as to what is amiss when he sees a group of feeble-minded persons. As far as actual measurements are concerned, feeble-mindedness shows itself, above all, in abnormal smallness of the skull. From time to time, indeed, we see an idiot with a large head; but, on the other hand, persons with well-marked microcephaly are invariably feeble-minded. According to Ziehen, we must suspect feeble-mindedness when we find that the circumference of the head is less than 52 centimetres.

The name of *schizophrenia* or *dementia praecox* is given to the commonest form of insanity. The trouble usually begins in the second or third decade of life, but sometimes later. There are various forms of the disorder; common to them all, however, is an extensive failure of the affective or of the voluntary life. In most cases, too, there is a more or less marked failure of the intellectual powers.

The patients are subject to purposeless impulses and purposeless inhibitions. Irregular impulses to movement may prove fatal; but in other cases there is an irremediable hebetude and a failure of will power, as we see in the severest form of the disease, which is known as *catatonia*. In yet other instances, the malady may from the outset take the form of a simple progressive mental decay, and is then known as *dementia simplex*. *Hebephrenia* is characterised by a peculiar desultoriness of thought. In other cases, illusion and unsystematised delusions may dominate the picture, and we then speak of *dementia paranoides*. If, on the other hand, the delusions are systematised and persistent, we speak of *paraphrenia*. In most cases schizophrenia has a somewhat paroxysmal course, there being sudden exacerbations of the symptoms, with intervening periods of improvement, until at long last the malady enters into a stationary phase.

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[1] It has also to be remembered that feeble-minded women are much more likely to be the mothers, than feeble-minded men are likely to be the fathers, of illegitimate children.—TRANSLATORS' NOTE.

No other cause than hereditary predisposition has yet been discovered in the case of schizophrenia. E. Rüdin,[1] who was the first to study the heredity of mental disorders from a genuinely modern outlook, after examining 701 families in which cases of schizophrenia had occurred, found that among the brothers and sisters of patients both of whose parents were healthy, 4·5 % likewise suffered from schizophrenia. When one of the parents was a schizophrene, the percentage of schizophrenes among the brothers and sisters of schizophrenes was 6·2 % (34 families). Among half-brothers and half-sisters, only 0·6 % were schizophrenes. These facts speak, moreover, in favour of recessiveness of the taint. So does the circumstance that, among 700 cases, Rüdin found that in 14 instances (that is to say in 2 ± 0·5 %) the patients were the offspring of cousins. In view of the great frequency of schizophrenia, by which from ½ to 1 % of all births become affected, we should not, even if the heredity be recessive, expect to find in the ancestry of the patients much more than the average frequency of cousin marriages—the average frequency in the general population being about 1 %. H. Hoffmann[2] has found that, among 150 children of schizophrenes, 13 to 15 (that is to say from 8·6 to 10 %) suffered from mental disorder of a schizophrenic type. He shares Rüdin's opinion that a doubly recessive determination must be at work, that is to say that two pairs of abnormal genes must be present, if dementia praecox is to result.

Persons suffering from a schizophrenic taint usually show slight mental anomalies before the actual outbreak of the disease. We note in them a certain blunting of the affective and voluntary life. On the other hand, in the families to which persons suffering from schizophrenia belong, we can usually detect the presence of "schizoid" psychopaths, who may never become affected, and indeed usually do not become affected, with well-marked schizophrenia. Most of the persons who are commonly spoken of as "eccentric", persons who are shy, unsociable, self-centred, reserved, cold, and devoid of a sense of humour, belong to this category. The Bavarians have a way of saying that some one "is spinning", and the term applies aptly to these minor degrees of schizophrenia. A schizophrenic condition may well be compared with spinning after the old style, the spinner being one who spends hour after hour twisting a thread, repeating the same movements over and over again and having no contact with the outer world. For the schizoid psychopaths who wrap themselves up in their own qualities, a comparison to a caterpillar which invests itself in a cocoon of its own spinning may be regarded as apter still.

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[1] Zur Vererbung und Neuentstehung der Dementia praecox, Springer, Berlin, 1916.

[2] Die Nachkommenschaft bei endogenen Psychosen, Springer, Berlin, 1921.

Rüdin, Hoffmann, W. Strohmeyer,[1] and others have supposed that heredity factors competent when acting homozygotically to produce schizophrenia may, perhaps, when acting heterozygotically, manifest their effects by the production of schizoid psychopaths. Such factors would then have to be regarded as intermediate. If we confine our attention to well-marked cases of mental disorder, and reckon as healthy the mere psychopaths in a kinship, the heredity of the illness would seem to be recessive. But if, on the other hand, we take into account the psychopaths as well as the persons suffering from developed mental disorder, the heredity of the malady will be seen to be dominant. Decisively in favour of the hypothesis that the factor behaves in an intermediate fashion is the circumstance that when two psychopaths marry one another they often have schizophrenic children. For the nonce, we cannot ascertain whether in every case of schizophrenia both parents have been schizoid, as they ought to have been if the foregoing hypothesis be sound. In like manner, it is still uncertain whether all the children of a schizophrenic parent are really schizoid psychopaths; but certainly most of them seem to be. If both parents were schizophrenes, then, in accordance with the notion of a recessive and an intermediate heredity, we should have reason to expect that all the children would be schizophrenes, provided that there was a biological identity in the heredity of the malady in both parents. Certain families have been described in which this expectation would seem to have been fulfilled; but in other families the facts do not appear to fit the theory, perhaps because, in these latter instances, there was no biological identity between the parental morbific heredities. Relevant family histories have been published by E. Kahn.[1]

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[1] Zur Genealogie der Schizophrenie und des Schizoids, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. XCV, 1925.

Meanwhile we have to admit that the problem of the heredity of schizophrenia has been no more than partially solved. As Siemens has pointed out, the systematic study of twins will enable us to decide whether external causes are of any importance in the causation of this disease. In most cases where schizophrenia has been found in twins, there is a lack of satisfactory evidence as to whether the twins were or were not mon-oval. Schulte,[2] however, reports a case in which twin brothers who could certainly be regarded as mon-oval suffered in like manner from catatonia. As far as it has gone, the study of schizophrenia in twins has tended to confirm the impression that external influences have little or no importance in the production of the disease, and modify its course very little.

W. Strohmeyer has been able to trace the schizoid or schizophrenic taint in the history of the German reigning families for centuries.[3] The ruling families are particularly suitable material for such studies because we are so exceptionally well informed regarding them for many generations back. We find that among the offspring of William the Younger of Brunswick-Lüneburg,[4] who obviously suffered from schizophrenia, there were numerous schizoid psychopaths, such as George I, king of Great Britain and elector of Hanover, and his son George II, also Frederick William I of Prussia and his son Frederick II. The example of the two last named shows that the schizoid taint does not necessarily entail disastrous results. In Louis II and Otto I, kings of Bavaria, the morbid taint was manifestly derived from both parents. Otto I suffered from typical schizophrenia (dementia simplex). Louis II is often said to have suffered from paronoia. Seeing, however, that in him there ensued auditory and visual hallucinations as well as failure of the reasoning powers, we can confidently diagnose the existence of schizophrenia (in the form of paraphrenia). Fig. 148 shows how from William the Younger different lines proceeded, and how the morbid taint could be transmitted in both of these until after nine or ten generations the trends could reunite. In this instance there is likewise a history of psychopathic disorders in the parental and grandparental generations. We cannot trace the morbid heredity farther back than this, but we have no right to assume that it did not exist.

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[1] Schizoid und Schizophrenie im Erbgang, Springer, Berlin, 1923.

[2] Ueber Katatonie bei Zwillingen, "Zentralblatt für die gesamte Neurologie und Psychiatrie", vol. XXXIII, 1923, p. 170.

[3] Op. cit.; also, Die Ahnentafel der Könige Ludwig II und Otto I von Bayern, "Archiv für Rassen- und Gesellschaftsbiologie, 1910, no. 1.

[4] I am indebted to Dr. Friedrich Lehmann of Munich for information regarding this potentate's descendants.

In Fig. 149 I publish an extract from a genealogical tree communicated to me by Hanhart. This tree comports well with the assumption that certain morbific heredity factors can produce heterozygotic schizoid psychopathy, while homozygotic morbific heredity factors can produce schizophrenia. These data come from one of the regions in Switzerland where inbreeding prevails. This hypothesis is likewise supported by the results of a family enquiry made by J. Lange.[1]

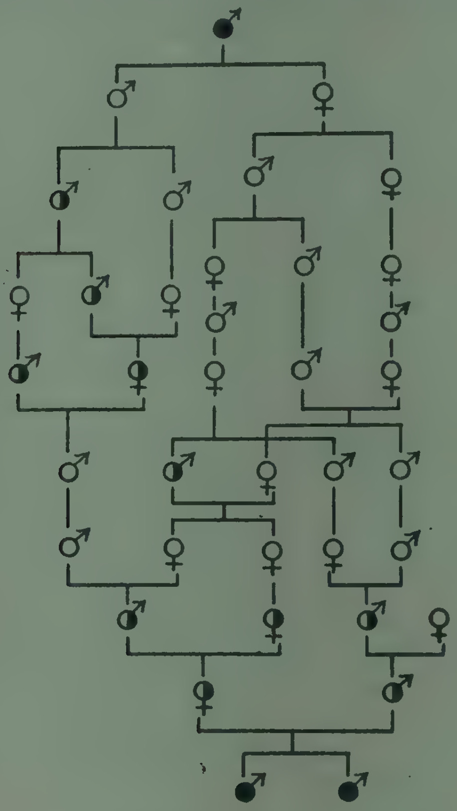

FIG. 148.

The descent of the Bavarian kings Louis II (born 1845) and Otto I (born 1848) from William the Younger of Brunswick-Lüneburg (born 1535).

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[1] Genealogische Untersuchungen an einer Bauernsippschaft, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. XCVII, 1925, nos. 3 and 4.

Among the Jews, cases of mental disorder are from twice to thrice as common as in the Gentile population. Since we are here concerned chiefly with instances of schizophrenia, we see herein additional evidence of the cooperation of recessive taints.

For the rest, it is altogether improbable that the group of schizophrenias, considered from the outlook of heredity, represents a biological unit. Just as we saw when studying the muscular atrophies, there are probably several different kinds of schizophrenia, hereditarily distinct, and merely classed together because they present clinical resemblances. E. Kretschmer[1] is far from wishing to maintain that the schizophrenic or schizoid forms of mental disorders constitute a biological unity. Again, E. Bleuler,[2] who originated the name schizophrenia, regards the group of schizophrenias as comprising a number of biologically distinct anomalies. The splitting-up of this group into its diverse biological elements must, however, be left to future investigators.

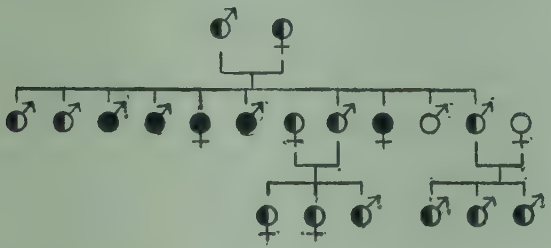

FIG. 149.
Schizophrenia and schizoid psychopathy (extract), after Hanhart.

The paranoid mental disorders would seem to be closely related to the schizophrenias. One in whom a persistent complex of delusions arises without any marked disturbance of clarity of thought in other respects, is said to suffer from *paranoia*. There are various intermediate grades between developed paranoia and mental health. Paranoid psychopaths are characterised by a peculiar lack of confidence in the persons and things of their environment, and this suspiciousness is apt to be associated with a more or less conspicuously exaggerated self-esteem. They feel that they are misunderstood, surrounded by foes, wronged, and slighted; they complain that they are not treated with due consideration. According to Economo,[1] maniacal litigiousness (in which there may or may not be a core of reasonable grounds for the litigiousness) belongs biologically to the group of schizophrenias. Studying the family history of litigious persons, Economo found that among their descendants from one fourth to one third became affected by schizophrenia, and that barely one third were mentally healthy, the residuum consisting of psychopaths (schizoids). J. Lange,[2] on the other hand, contends that the predisposition to paranoia or to paranoid psychopathy is biologically distinct from the predisposition to schizophrenia. If he is right, we must explain the frequent coincidence of cases of paranoia and cases of schizophrenia in the same family as the outcome of selection (the tendency of various kinds of psychopaths to intermarry).

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[1] Körperbau und Charakter, third edition, Springer, Berlin, 1925; English translation by W. J. H. Sprott, Physique and Character, Kegan Paul, London, 1925.

[2] Lehrbuch der Psychiatrie, Springer, Berlin.

Indubitably, hereditary predisposition is an important contributory factor of *senile mental enfeeblement.* It is a familiar fact that the age at which senile mental enfeeblement begins varies much from person to person, and that there are great differences in the severity of this kind of mental disorder. When I was a student I was acquainted with two brothers about eighty years old for whom I was hardly a match as a chess player, although I had had plenty of experience of the game. Most people, however, have passed the climax of their mental powers by the time they are forty. Moebius holds that those who in old age suffer from well-marked mental disorder have hardly ever been wholly normal. Instances of dementia praecox are comparatively often found among the offspring of persons who in old age have come to suffer from *dementia senilis,* so that the existence of a connexion between the two diseases may be presumed. Perhaps schizoid psychopathy indicates a predisposition to senile dementia. It would also seem that psychopathic taints which have been very little manifest during the prime of life may become conspicuous in old age through a decay in the power of self-criticism. It is probably true that the onset of senility can be accelerated by severe mental and bodily labour, by anxiety and excitement, by the abuse of intoxicants, etc.; but the hereditary equipment obviously plays a still more important part here. It is very difficult to draw up trustworthy family trees illustrating the hereditary causation of senility, for most persons die prematurely, and concerning the living offspring of those who are or have been affected with senile feeble-mindedness we do not know what their mental condition will be in old age.

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[1] Ueber den Wert der genealogischen Forschung für die Einteilung der Psychosen, "Münchener Medizinische Wocnenschrift", 1922.

[2] Ueber die Paranoia und die paranoische Veranlagung, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. XCIV, 1924.

To the group of hereditarily determined dementias we must also assign what is known as *amaurotic idiocy*. (Amaurosis is a general name for blindness due to disease of the optic nerves or the retina.) This is a grave progressive malady which appears during the first or second year of life in children which to outward seeming were previously normal. After increasing mental failure, convulsions, paralysis of the limbs, and blindness have ensued, it ultimately proves fatal. This course shows that the malady has nothing to do with other forms of idiocy, where the feeble-mindedness is inborn and not progressive, but is akin, rather, to the hereditary atrophy of the yellow spot described in the subsection on diseases of the eye (see above, p. 250). It might well be termed dementia amauroticans. Noteworthy is the fact that this disease, whose heredity seems to be simply recessive, occurs almost exclusively in Jewish families.

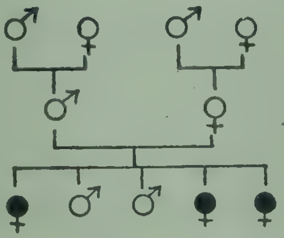

FIG. 150.
Amaurotic idiocy, after Starck.[1]

Similar to amaurotic idiocy are certain other rare diseases whose heredity appears to be likewise simply recessive, and which first manifest themselves in the later years of childhood. Thus we find families in which several children during the second half of the first decade of life exhibit a decline in the intellectual powers and at the same time suffer from impairment of vision, until at last complete dementia ensues.[2]

This affection, however, though in other respects it resembles amaurotic idiocy, contrasts with the latter by occurring in Gentile and not in Jewish families.

Hereditary predisposition is of little importance in the causation of *dementia paralytica* (commonly spoken of as *general paralysis of the*

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[1] Zur Kasuistik der familiären amaurotischen Idiotie, "Monatsschrift für Kinderheilkunde", vol. XVIII, no. 2.

[2] W. Spielmeyer, Ueber eine besondere Form von familiärer amaurotischer Idiotie, "Neurologisches Zentralblatt", 1906, p. 51.

insane, and sometimes though less aptly termed "softening of the brain"). Here we have to do with a special manifestation of syphilis, a destruction of brain tissue which usually proves fatal in from one to two years. The hereditary equipment of the syphilitic virus seems to have more to do than the hereditary equipment of the infected person in deciding whether this individual or that who suffers from syphilis becomes a general paralytic; but of even more importance is the way in which the syphilis is treated. To this matter we shall return in the sequel to the present work, when the problem of the increase in mental disorder will come under consideration. We find, however, that the mental symptoms of paralytic dementia are coloured by the individual predisposition of the patient. In schizoid families, for instance, the manifestations remind us of the schizophrenic types of insanity, whereas in cycloid families they remind us of the manic-depressive types.[1] (The term "manic-depressive" will be explained below on p. 444.)

The name of *epilepsy* is given to conditions of abnormal susceptibility of the cerebral cortex, usually characterised by attacks of unconsciousness accompanied by peculiar convulsions. The clinical picture of epilepsy can also result from the working of external causes, such as injury to the brain or (in childhood) acute brain infections. When there is no obvious external cause, we speak of the disease as "idiopathic" or "genuine" epilepsy. In severe cases, epilepsy usually results in dementia, and then on postmortem examination we find atrophy of the cerebral cortex.

A considerable proportion of all cases of epilepsy are supposed to be due to the abuse of alcohol (alcoholic epilepsy). The predisposition to epilepsy is also supposed to manifest itself at times in the form of other kinds of mental disorder, as, for instance, in paroxysms of uncontrollable restlessness or in paroxysms of mental confusion without convulsions (sometimes called "twilit mental states", or "crepuscular states"). It is obvious, however, that many authorities have inclined to extend the concept of epilepsy too widely, subsuming under this term various conditions which really have nothing at all to do with it. Kurt Schneider[2] refuses to admit that there are epileptoid psychopaths.

A notable proportion of the children of epileptics die in early life from "convulsions". On the other hand, there are many epileptics who in the course of a long life have only one fit or no more than a very few fits, and this leads us to suspect that there may be a great many persons affected with the epileptic taint who never have any fits at all. Obviously if this be so, the tracing of the heredity of epilepsy becomes very difficult.

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[1] F. W. Kalb, Beiträge zur Belastungsfrage bei Paralyse, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. LXIII, 1916, p. 391.

[2] Die psychopathischen Persönlichkeiten, contributed to Aschaffenburg's Handbuch der Psychiatrie, Leipzig and Vienna, 1923.

About 10 % of all epileptics are known to have had an epileptic parent, and among the children of epileptics about 10 % are epileptic. Among the collateral relatives of epileptics, there is a still larger proportion of sufferers from the disease. According to E. Rüdin[1] there are recessive hereditary factors capable of causing epilepsy, but perhaps in less common instances the disease may be produced by factors whose heredity is dominant. It is a remarkable fact that consanguineous marriages do not appear to be especially common among the parents of epileptics. In any case, the clinical picture of epilepsy is not a biological unity. Henceforward, therefore, investigators must try to distinguish between the elementary sub-varieties of epilepsy.

One of these elementary sub-varieties of genuine epilepsy is *myoclonus epilepsy*, which is characterised by peculiar paroxysms of muscular twitchings wherein each individual twitch is so brief that the intended movements become impossible. In this malady the epileptic attacks usually come on during the night. Ultimately the disease leads to dementia. H. Lundborg[2] observed 17 cases among the children of consanguineous parents. The ratio between affected and unaffected persons in these children corresponds to the theory that the heredity is recessive, so that this theory can now be regarded as established.

All the 17 cases of myoclonus epilepsy known to have occurred in Sweden were found by Lundborg to be the descendants of the same ancestral pair in the eighteenth century. Other cases of the disease are known to have occurred in the Baltic area, and perhaps these are genealogically connected with the Swedish cases.

In Fig. 152 I reproduce from Weeks[3] a family tree which manifests the working of an obviously dominant heredity factor leading to epilepsy.

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[1] Der gegenwärtige Stand der Epilepsieforschung, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. LXXXIX, 1924, nos. 1 to 3.

[2] Medizinisch-biologische Familienforschungen, etc., Fischer, Jena, 1913.

[3] Communicated to the Second International Congress for Racial

Davenport and Weeks[1] have almost always found feeble-minded-

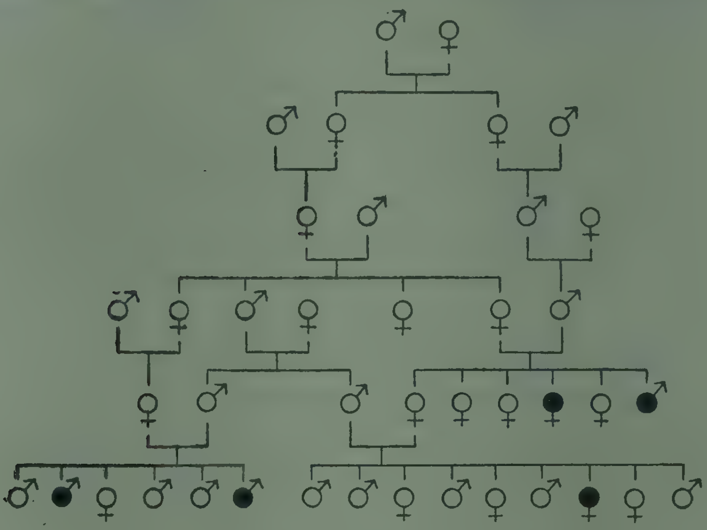

FIG. 151.
Myoclonus epilepsy, after Lundborg (extract).

ness in the families of epileptics, especially among the brothers and sisters of the epileptic patients. Epileptics are themselves apt to be feeble-minded. It would seem, then, that there must be heredity factors competent to produce, on the one hand feeble-mindedness, and on the other hand epilepsy. Still, we must remember that in certain kinships two distinct heredity factors, one a factor of feeble-mindedness and the other a factor of epilepsy, may be conjoined because (as I have already more than once insisted) mentally defective persons are so apt to mate with mentally defective persons. One such family tree is reproduced in Fig. 153. Especially noteworthy in it is the fact that in one branch of the family all the seven children of one generation suffered from epilepsy.[2]

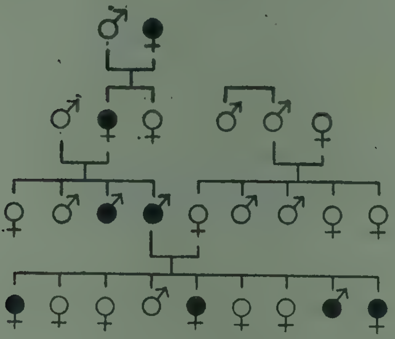

FIG. 152.
Epilepsy, after Weeks.

Hygiene, held at New York in the year 1921, and reproduced in Eugenics, Genetics, and the Family, Williams & Wilkins, Baltimore, 1923.

[1] A First Study of Inheritance in Epilepsy, "Eugenics Record Office Bulletin", 1911, no. 4.

[2] Reported by H. H. Laughlin, Eugenic Sterilisation in the United States, Chicago, 1922.

Roemer[1] has described a kinship in which, together with epileptics, there were a great many psychopaths. He considers that the same heredity factor probably accounted for both morbid conditions. Hitherto epilepsy has seldom been observed in twins. Herrmann,[2] however, saw two mon-oval twins who both suffered from epilepsy and in whom the disorder ran exactly the same course. In Jews epilepsy is less common than among Gentiles.

Among epileptics we find more than twice as many males as females, even during the first two decades of life, when the effects of alcohol and other noxious influences cannot be invoked to account for the difference. From this we may infer the probability that there may also be recessive sex-linked factors which play a part in the causation of epilepsy.

To epilepsy similar considerations apply as to the inflammatory diathesis (see above, p. 326). Potentialities for the onset of an epileptic paroxysm are already provided as parts of a healthy hereditary equipment, but in normal persons epileptic paroxysms can only be induced by powerful external noxious influences, such as injuries to the brain. When there is abnormal susceptibility, slighter causes acting from without, such as the abuse of alcohol, may give rise to epilepsy. Finally, persons with the epileptic diathesis are those in whom the presence of certain heredity factors has determined so strong a susceptibility that fits ensue even as an outcome of the ordinary influences of everyday life. When one gene suffices to activate in this way the tendency to epileptic paroxysms,

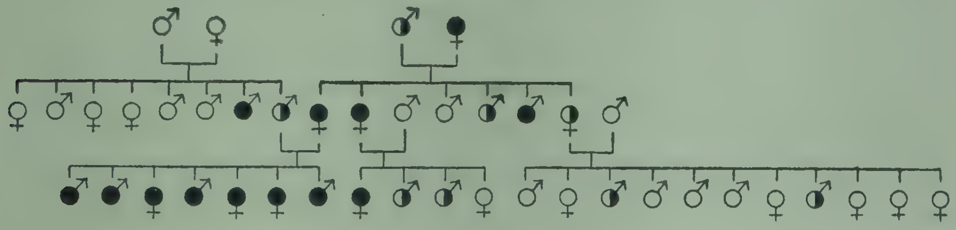

FIG. 153.

Epilepsy and feeble-mindedness, after Weeks and Laughlin (extract). The individuals denoted by half-black circles were feeble-minded.

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[1] Zur Symptomatologie und Genealogie der psychischen Epilepsie und der epileptischen Anlage, "Allgem. Zeitschr. f. Psychiatrie", vol. LXVII, 1910, p. 588.

[2] Epileptische Anfälle mit typischer, vollständig gleichartiger Symptomatologie bei Zwillingen, "Medizinische Klinik", 1919.

the course of the morbid heredity is dominant; but if, on the other hand, the cooperation of two similar genes is requisite to cause this activation, then the morbid heredity is recessive. In epilepsy, recessive heredity would seem to be commoner than dominant.

As regards the true insanities, we find that the importance of hereditary predisposition is most clearly marked in what is termed *manic-depressive insanity* (and would perhaps be better called manic-melancholic insanity). This name is given to a group of mental disorders which are characterised by more or less severe disturbances of mood. One of these disturbances is melancholia, in which all the processes of the mental life are coloured by profound sadness; another is mania, wherein the mental life is morbidly disturbed by uncontrollable and cheerful excitement; the third leading form is that which is known as circular or periodic insanity, wherein the patient's mental state fluctuates between the maniacal and the melancholic passing from one to the other by way of the normal. Nevertheless, when the predominant picture is one of simple melancholia or simple mania, we find that the patient tends after a shorter or a longer time to pass back for a while into a normal or an approximately normal state. Usually, however, in such patients, we find that, even when their minds are comparatively healthy, lesser anomalies can be detected pointing to mania or to melancholia as the case may be. In yet other persons we note slight manifestations of the manic-depressive state, but no mental illness that we are entitled to call mania or melancholia ever arises. To this category belong those who in ordinary life are known as "sensitive" and "moody" persons.

If we wish to have one simple name for the whole group of manic-melancholic mental disorders, we can use the term *parathymia* (the root "thym" denoting the mind, the moods, or the spirits; and the

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[1] At about the same time as when, in the first edition of this book, I proposed to apply the name parathymia to the whole group of manic-depressive disorders, the psychiatrist Meggendorfer began to use the term in an entirely different sense, applying it to "temporary" cases belonging to the group of schizophrenias and manifesting themselves in the form of "moral insanity". (F. Meggendorfer, Klinische und genealogische

word "parathymia" thus signifying disorder of mood).[1] Conditions of maniacal excitement can be spoken of as hyperthymic, and conditions of melancholic depression can be termed hypothymic.

The lesser anomalies of this group, those which do not transcend the domain of psychopathy and never reach the intensity of developed insanity, are by Kretschmer spoken of as "cycloid", and he speaks of the whole tendency to such alternations of mood as "*cyclothymia*".

Whereas the normal individual finds it impossible to put himself imaginatively in the place of a person suffering from schizophrenia, we can all of us in great measure understand the alternations of mood of the manic-depressive patient. We are all of us in "high spirits" at one time, and in "low spirits" at another, but usually for some good reason, such as success or ill-success in love. The maniac is one who is in fantastically high spirits, and the melancholic is one who is in fantastically low spirits—and in either case without good cause. In hyperthymic states, the person concerned has a surge of happy feelings, is intensely self-confident, enterprising, and intellectually active. In its morbid extremes, however, such a mood is unfavourable to self-preservation, for it leads to hasty and ill-considered actions.

In many cases we can trace the heredity of manic-melancholic mental disorders through several generations. It may be assumed, therefore, that some at least of the factors of this group have a dominant heredity. The data collected by all those investigators (such as E. Rüdin,[1] H. Hoffmann,[2] and A. Wimmer[3]) who have worked with trustworthy methods harmonise with this view. In a material comprising 650 cases of manic-melancholic mental disorder, Rüdin, indeed, was able to find evidence of pronounced mental disorder in the parents in only one fourth of the patients. In 566 series, among the brothers and sisters of manic-melancholic patients, only 7·4 % were likewise insane. But in cases in which one of the

Untersuchungen über "Moral Insanity", "Zeitschrift für die gesamte Neurologie und Psychiatrie", 1921.) I doubt whether any such groups as Meggendorfer speaks of can be clearly circumscribed. Even apart from this, it seems to me injudicious to use the root "thym" to denote any of the forms of schizophrenia.

[1] Ueber Vererbung geistiger Störungen, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. LXXXI, 1923, nos. 3 and 4.

[2] Vererbung und Seelenleben, Springer, Berlin, 1922.

[3] Sur la transmission héréditaire des maladies mentales, "L'encéphale", seventeenth year of issue, 1922, p. 129.

parents of the patient was insane, 24·6 % of the brothers and sisters were insane. Consanguinity of the first degree was found in only 0·9 % of the parents, no more often than among the general population, and this is opposed to the idea that recessive heredity factors play a part in the production of the disease. Among the relatives of manic-melancholic patients we usually find what are called cycloid psychopaths. Those who in the end actually become maniacal or melancholic have usually ere this exhibited signs of a cycloid predisposition. It would seem, then, that most of the factors which can determine manic-melancholic disturbances are dominant, and that in persons thus predisposed actual mania or melancholia is caused by influences which still remain obscure.

The fact that among patients suffering from maniacal and melancholic conditions there are nearly twice as many women as men does not appear to depend upon a sex-linked heredity, but upon the circumstance that women in general are more prone than men to be affected by vacillations of moods. In conformity with this we find that the tendency of the female form as compared with the male is to be rounded, and we know that the "pyknic" type is correlated with the cycloid temperament. Speaking generally it would seem that the onset of manic and melancholic states depends rather upon disturbances of the endocrine balance ("internal secretion") than upon primary disorders of the brain. This theory is supported by the periodic course of the maladies in question, and also by the fact that there is no evidence of irreparable changes in the tissues.

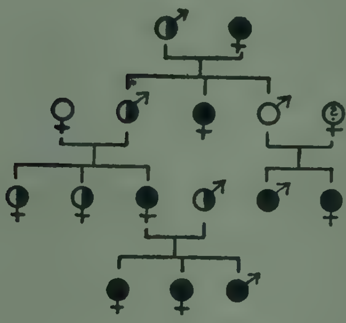

FIG. 154.

Manic-melancholic disorders, after Hoffmann.
Half-white circles denote hyperthymic psychopaths.

In Jews manic and melancholic disorders are commoner than in Gentiles.[1]

A considerable proportion of persons predisposed to manic-melancholic disorders, and especially of those in whom melancholia is predominant, die by suicide. The tendency to commit suicide has often been observed to run in families, and there is no evidence that the force of example plays an important part here.

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[1] J. Lange, Ueber manisch-depressives Irresein bei Juden, "Münchener Medizinische Wochenschrift", 1921.

In Fig. 155 I reproduce a relevant family tree published by F. W. Bremer.[1]

The hereditary equipment is of even more importance in the production of those lesser mental anomalies which are termed *psychopathies* than it is in the production of the developed forms of insanity. Whereas in the case of some kinds of insanity external noxious influences are decisive in their production (for instance, general paralysis of the insane), such influences have hardly anything to do with the causation of the psychopathies. Somewhat illogically, it is customary to apply the term "psychopathies" exclusively to anomalies of the affective and the impulsive life, and not to place cases of weakness of the understanding in this group.

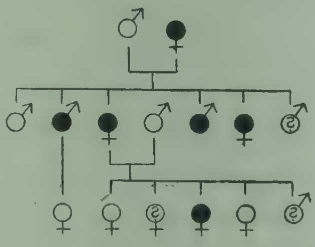

Fig. 155.
Suicide, after Bremer (extract).

I should prefer to class among psychopathies all the lesser mental anomalies, including those of the intelligence. I regard as untenable a distinction of the insanities as "processes" from the psychopathies as "states", the only suitable ground for distinguishing a psychopathy from an insanity being that the former is a slight and the latter a very serious disorder. There are numerous grades of transition between psychopathy and health. For this reason, the point when psychopathy may be said to begin is somewhat arbitrarily determined, and the number of cases of psychopathy cannot be unambiguously ascertained. Complete mental health is doubtless as rare as perfect bodily health. But even if we restrict the application of the term psychopathy to such mental conditions as obviously result in a below-average capacity for self-preservation, we find that a very considerable fraction of the population must be classed as psychopathic—perhaps as much as a tenth. F. Prinzing,[2] a noted authority on medical statistics, holds that about 10 % of the population are mentally below par.

Alienists have hitherto found it just as difficult to formulate a satisfactory classification of the various psychopathies as of the developed insanities. For the most part we have to do here with manifestations of taints which have already been considered in connexion with the insanities, namely the schizoid, the paranoid, the epileptoid, and the cycloid temperament or predisposition. These are respectively associated with the kindred psychoses, so that the biology of each psychopathy and the biology of the allied insanity must be jointly considered.

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[1] Zur Vererbung der Selbstmordneigung, "Archiv für Psychiatrie", vol. LXXIII, 1925, nos. 2 to 4.

[2] Die statistischen Grundlagen der sozialen Hygiene, contributed to Gottstein's Handbuch der sozialen Hygiene, Berlin, 1925.

In ordinary social life, the psychopathies play a more important part than do the developed insanities; for, whereas in most cases the insane are speedily excluded from social life, psychopaths remain in society and influence it very markedly in all sorts of ways. There are various sects and political organisations a large proportion of whose members are psychopaths, and there are numerous "movements" which are largely directed by psychopaths.[1] Thus in times of social disturbance psychopaths may do a great deal of mischief, though sometimes of course they may do good.

The *hysterical temperament* is of exceptional importance both in private and in social life, and for this reason I shall discuss it at greater length than other mental anomalies. In my opinion an understanding of the mechanism of hysteria is absolutely indispensable, not only to the physician, but also to the educator (especially the popular educator), the statesman, the economist, the historian, and not least the racial hygienist.

When we speak of hysteria, we think primarily of certain ostensibly physical conditions for which however no bodily foundation can be detected and which may suddenly change their manifestations or vanish without leaving a trace. First we have hysterical paralyses of the limbs, anaesthesia of circumscribed areas, "rheumatic" and other pains, blindness, deafness, dumbness, syncopal attacks, and a medley of other symptoms, which may all come within the domain of hysteria. Very characteristic are peculiar convulsive seizures with clouding of the consciousness which to outward seeming may resemble epileptic fits. I should define hysteria as a more or less unconscious and involuntary mimicry of various other diseases.

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[1] Cf. H. Hentig, Schutz der Republik gegen Geisteskranke, "Deutsche Strafrechtszeitung", vol. IX, 1922, p. 291.

It is not necessary for the hysterical patient to have seen a case of the disease he is imitating. He produces a picture of what he imagines the disease to be like. We may compare hysteria to mimicry in the animal and vegetable kingdoms. An animal which alarms an assailant by mimicking the behaviour of some venomous or otherwise dangerous creature, presumably does not know that it is imitating. When a hen grouse shams a broken wing in order to distract attention from her brood, she acts (one may suppose) instinctively and unconsciously. The same remark applies to the "shamming dead" of many creatures, especially insects.

The appearance of individual hysterical manifestations is the expression of a strong desire to obtain an object or gain an end by a hysterical person who believes, more or less unconsciously, that in this way he will be helped to achieve his purpose. During the war, for instance, the wish to get away from the enemy fire and to return home led to the onset of all kinds of hysterical symptoms, one of the most frequent of which was the occurrence of persistent tremors and twitchings.[1] In "railway-accident hysteria", for instance, the person who has been in an accident wishes for compensation, and the wish, acting through the unconscious, evokes symptoms appropriate to the attainment of this end. But what distinguishes hysteria from simulation is that in hysteria the association between the symptoms and the longing is plunged more or less deeply in the unconscious. In everyday life the cause of hysterical symptoms is often the unconscious longing to evade disagreeable duties, or to arouse sympathy, or to make oneself interesting, or to put some one else in the wrong (some one who can be blamed for the onset of the illness). Epileptoid symptoms are so common in hysterical patients because the epileptic fit always makes a great impression upon the laity. For a long time, indeed, epilepsy was known as "morbus sacer", the "holy disease". When the hysterical patient is in strong convulsions, anger which may have been felt against him for shirking his duties is apt to be transformed into sympathy or into superstitious dread. Another impressive illness which hysterics are fond of imitating is chorea. But the hysterical patient will imitate mental morbid conditions as well as bodily ones, desiring to arouse sympathy by the mimicry of melancholic states, or to inspire alarm by the mimicry of states of mental excitement. A hysterical person who is being prosecuted for some offence against the law will be apt to show signs of weakness of the understanding or of the memory if this is likely to redound to his advantage. The illness is willed as a means for the attainment of a strongly desired end.

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[1] Kretchmer has described with a fine sympathetic insight the way in which such conditions arise. See Ueber Hysterie, Thieme, Leipzig, 1923.

Whereas the actual onset of hysterical symptoms depends upon external causes which arise from desires, its constitutional foundation is a hereditary proclivity to have ideas, judgments, feelings, and even sensations, determined by more or less unconscious wishes. This abnormal facility to be influenced by desires may show itself in other ways besides the imitation of various forms of illness. Far more often, indeed, it shows itself negatively in the repression of disagreeable ideas or memories, which are driven out of consciousness; or it shows itself positively in the production of all kinds of fancies or wish-illusions.[1] Thus, it often comes to pass that persons endowed with the hysterical temperament fallaciously suppose one or more persons of the other sex to be in love with them. The flight away from fact and into a world of illusions is characteristic of the hysterical proclivity to be influenced by wishes.

Since I regard it as desirable to restrict the term hysteria to an involuntary mimicry of disease (and this restriction is in conformity with the best medical usage), I have proposed to coin the word *orgoristia* as a general term for the underlying morbid facility to be influenced by wishes, which can manifest itself in other ways as well as in the production of morbid symptoms. This word, however, not having been coined by a psychiatrist, has not caught on, and henceforward therefore in this book I shall speak simply of the hysterical temperament.

Women with the hysterical temperament try to get their own way by making a parade of their sufferings, and by an unexpressed threat that if they are thwarted these sufferings will last for ever. Tears and a distressful mien are with them favourite expedients. Fainting fits, on the other hand, are less in fashion than they used to be. Of course these means are only effective when used to influence persons who are bound to the hysteric by love or duty and who themselves suffer when the hysteric suffers. For the rest, it is especially easy for the hysteric to get his way in a moral environment where suffering is considered meritorious, and where the view prevails that pain and asceticism are useful for their own sake.

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[1] Contrary to general linguistic usage, the word "illusion" has been arbitrarily introduced into psychiatry with the signification of a false sensual perception. I do not here use it in that sense.

Hysterical mimicry is a kind of parasitism which flourishes in the soil of the ascetic doctrine of the value and morality of sympathy.

I need hardly say that we must avoid the extreme of accounting morbid every determination by a wish. The healthy human being is apt to believe what he wishes, but experience and logical thought set bounds to this determinability by wishes. Manifestly some degree of determinability by wishes favours self-preservation and species-preservation. Thanks to mental determinability by wishes, opinions and voluntary trends which have been found favourable to the preservation of a community, become elements in the volition of the individual members of that community. This is an advantage to the community in the struggle for existence. Thus during the world war, the illusion that Germany was alone responsible for bringing the conflict about and that every German was a scoundrel, unquestionably contributed to promote in our enemies the power to hold out to the end and strengthened them for the victory. We must not regard such illusions as necessarily the outcome of morbid predisposition, although hysterically predisposed persons are most inclined to entertain them. This applies to other hysterical convictions which are very widely diffused, such as that every Jew is a rascal—or that, according as the outlook of the accuser varies, every freemason, every Jesuit, every bourgeois, every socialist is a rascal. The extent to which such facile generalisations will gain possession of any one's mind depends upon the ratio between his determinability by wishes, on the one hand, and his powers of insight, on the other. Certain persons who are not gifted with much intelligence can easily be persuaded, even though they are free from hysterical taint, that a golden age of happiness and justice would ensue if only all the Jews could be expelled from the country, or that the introduction of a communist economic order would bring universal happiness and that thenceforward all men would be good and noble. But when comparatively gifted persons become inspired with such illusions, they must be presumed to be morbidly predisposed.

It would be an exaggeration to regard every involuntary imitation of morbid conditions as the outcome of the hysterical temperament. During the years of development, especially in girls, we sometimes see hysterical manifestations which soon afterwards disappear for ever. Nor must we regard as a hysteric every one who bursts into tears on some appropriate occasion. In a sense, all of us are a trifle hysterical. Some degree of determinability by our wishes is normal. Such determinability is one of the foundations of love, morality, religion, and art.

It is a familiar experience that exceptional capacity for making effective suggestions to others is usually associated with a tendency to be strongly influenced by autosuggestion. Persons with a hysterical temperament may give far-reaching pledges to themselves and others, often without words, and without any clear notion of what they are doing. A demagogue who was not hysterical would have missed his vocation. The mass soul reacts like that of the hysterical individual. Sanity is promoted in the individual man by the recognition that others do not share his illusions. But when such illusions arise simultaneously in large numbers of persons under the influence of a common need or a common craving, the individual regards the agreement of the crowd as a confirmation and justification of his own illusions, and gives them free rein. Thus the wish illusions of the many reinforce one another, until ultimately all the barriers of inhibition are broken. One who adduces reasonable considerations is cried down, and is persecuted as an enemy of the people. The demagogue, on the other hand, who gives expression to the wish illusions of the many, is hailed with acclamation.

The determining wishes which give rise to hysterical reactions may be of very various kinds, according as circumstance and predisposition vary. In many cases these wishes are of an erotic nature, but psychoanalysts have tended to make far too much of the erotic motive. Only in the piping times of peace does the erotic urge stand in the foreground of human wishes. During the great war, the craving to live or the fear of death was the main cause of hysteria. The impulse to self-assertion rivals the sexual impulse and the impulse to self-preservation in the causation of hysterical symptoms. Persons endowed with the hysterical temperament use the most extraordinary means of attracting attention to themselves and of crowning themselves with a halo. Of late years, many cases have been recorded in which there has been "stigmatisation", a hysterical simulation of the wounds of Jesus—the person thus affected being usually an unmarried woman. Of course the great expectations which hysterically predisposed persons, with or without words, arouse in themselves or in other persons, are seldom fulfilled. The hysteric cannot get beyond the perpetual drafting of new plans, the use of magniloquent words, the making of fine gestures; but hysterics are never at a loss for an excuse, and they are masters in the art of hiding their weaknesses by shifting the blame on to others' shoulders. They simulate strength of character by acts of violence, by storms of temper, and by mulishness. The whole life of persons with the hysterical temperament has a theatrical flavour, and one might speak of their psychology as the psychology of "as if".

Hysterics, of course, cannot continue to impose upon persons to whom they are well known. That is why so many hysterical women regard themselves as "misunderstood", and why so many hysterical men are prophets not without honour save in their own country. When the hysterical temperament is associated with a malicious character (this association is comparatively infrequent), there results the well-known hysterical hatred for the hysteric's own associates or own country. Persons who do not share a hysteric's illusions are blamed because these illusions have not hitherto been realised.

Beyond question the hysterical temperament is mainly the outcome of the hereditary equipment. None the less, extensive researches concerning the heredity of hysteria have not hitherto been made by psychiatrists. The existence of this gap in the literature of genetics is in large measure to be accounted for by the difficulty in deciding the limits of the hysterical temperament. Genealogical sketches or other second-hand reports will seldom enable a physician who has not himself seen the person concerned to decide whether the hysterical temperament was or was not present. W. Medow[1] believes that he has been able in certain cases of hysteria to prove the homogeneous inheritance of the underlying constitution. In my own experience, hysterical predisposition tends to run so markedly in families as to produce the impression that the heredity is dominant. We must not assume, however, that all cases of hysteria are the outcome of the same heredity factor. In view of the existence of numerous transitions between the "hysteric" and the "normal", we must suppose that there is polymerism.

It is frequently asserted that hysteria is enormously more common in females than in males, some going so far as to say that there are ten female hysterics for every one male. Even if this be true as regards obvious manifestations of the hysterical temperament, we have no right to infer that the temperament itself is enormously commoner in women than in men. We have to remember that in peaceful times the mimicry of disease is not for men so apt a means as it is for women to get their own way. Experience during the great war has shown that the hysterical temperament is much commoner in males than used to be supposed.

The discovery that unconscious wishes are the determining causes of hysterical reactions is one for which we are mainly indebted to the Jewish psychiatrist Freud. In psychoanalysis, he has elaborated an effective (though not an exclusive) technique for the treatment of hysterical conditions. As soon as we are able to make a hysteric aware that the morbid manifestations from which he suffers are only the outcome of his own unconscious wishes, their foundation is withdrawn, and they disappear—but the underlying hysterical temperament remains. Freud and his disciples have failed to make due allowance for the importance of hereditary predisposition in hysteria. In its whole psychological structure, psychoanalysis is too exclusively concerned with causation by environmental influences, and especially by psychical experiences. Since it was found that the experiences of later life did not suffice to account for the hysterical temperament, which is obviously much stronger in some persons than in others, the Freudian school looked for evidence of some "psychical trauma", some shattering experience, in early youth; but inasmuch as people are apt to find what they look for, the Freudians can generally discover what they want in the form of some sexual or supposedly sexual experience. In actual fact, however, such experiences of childhood can, as a rule, be nothing more than the exciting causes which activate a predisposition which in any case would have been activated sooner or later by some other accidental cause. Thus the basic determinant lies much farther back, and is to be sought in the hereditary equipment.

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[1] Zur Erblichkeitsfrage in der Psychiatrie, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. XXVI, 1914, p. 493.

Since the temptation to a hysterical flight into illness is especially apt to arise in one who is not equal to the occasion, in one who is not competent for his task, very various bodily and mental weaknesses may contribute to originate hysterical manifestations; for instance, asthenia, cycloid depression, schizoid paralysis of the affective life, and, above all, neurasthenia. That accounts for the frequent amalgamation of hysterical with other psychopathic manifestations. In persons whose powers of judgment are at fault, hysterical states are commoner and cruder than in persons with more insight and endowed with a livelier faculty of self-criticism. That is why we so often encounter a combination of feeble-mindedness with hysteria. On the other hand, when hysterical mimicry does occur in highly gifted persons, it is of a subtler and more refined character. Seeing that, in addition to an undue liability to determination by wishes, several other constitutional trends collaborate in the production of hysterical symptoms, the hereditary basis of hysteria is to be regarded as polymeric.

We are often told that hysteria is especially common in Jews, and M. J. Gutmann[1] is one of those who take this view. Having had experience of thousands of Russian prisoners of war, I have been led to the conclusion that among the Ashkenazic Jews hysteria is less common than among the Russians. Strikingly common, on the other hand, among Jews are dread of illness (nosophobia) and hypochondria; and Lange tells us that in Jews melancholic conditions are unusually apt to be tinged with hypochondria. It would certainly seem to be a fact that where the male sex is concerned hysteria manifests itself more often in Jews than it does in the Teutonic races, the explanation perhaps being that Jews on the average have in respect both of body and of mind a less markedly developed masculine character.

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[1] Die Rasse- und Krankheitsfrage der Juden, Müller und Steinicke, Munich, 1920.

Neurasthenia manifests itself as an abnormally great liability to mental fatigue, and therewith to an undue irritability. Since we are concerned with a mental anomaly, the term *psychasthenia*, introduced by J. L. A. Koch,[1] and later unfortunately employed by Janet in somewhat too narrow a sense, would be apter. In contradistinction to the mental weakness known as feeble-mindedness, in psychasthenia we have to do with a mental weakness of a very different kind. The psychosthenic's mental alertness is in general above average, but his staying power is deficient. It is usual to draw a distinction between a constitutionally determined neurasthenia and a neurasthenia which has been acquired through over-exertion; but in both cases alike there can be no doubt that the condition is the joint outcome of hereditary predisposition and external causes. When the exertion necessitated by an ordinary occupation induces nervous exhaustion, the hereditary predisposition must be the most important factor; but when, on the other hand, neurasthenic symptoms arise as the outcome of exceptionally great exertion or exceptionally severe distress, we must consider these external causes to be the main factors.

As a rule the states of nervous exhaustion which are induced by overwork in a person with neurasthenic predisposition pass off speedily when the cause is removed. On the other hand it would seem that long-continued overwork may originate constitutional neurasthenia, or at any rate intensify it. We cannot but be astonished, however, at the enormous amount of mental work which many persons can do without becoming neurasthenic.

We can conceive the constitutional foundation of neurasthenia as being of either of two different kinds. In the first place, there may be too small a store of mental energy, meaning by "energy" the capacity to do work. In the second place, the mental energy, though present in normal quantities, may be expended too speedily; the substances out of which the central nervous system is supplied with energy for its work may be decomposed too readily, may burn up too fast. In such cases, drugs which retard the mental processes (the bromides, for instance) may give relief, whereas stimulating substances (like coffee) accelerate the onset of exhaustion. Mentally alert persons are more liable to neurasthenia than the mentally inert, so that in general we find neurasthenics to be more or less gifted. They are, to a degree, capable of brilliant achievements, but they lack staying power. Their performances may be compared to a firework display, which is brilliant while it lasts but is soon over. When there is bodily weakness, mental exertion more readily induces nervous exhaustion than it would if the physique were robust. For this reason, neurasthenics are often persons of an asthenic habit of body, but the association is by no means invariable.

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[1] Die psychopathischen Minderwertigkeiten, Ravensburg, 1891–1893.

A neurasthenic temperament seriously impairs the sense of wellbeing, especially when mental exertion leads to obstinate insomnia, which in its turn hinders recovery. For many a neurasthenic, life is a tragedy because, owing to his talents and his mental alertness, he feels himself called to tasks which, owing to his excessive liability to fatigue, he is never able to perform.

In about one third of all neurasthenics we can find evidence of psychopathic parentage. Had we fuller information regarding the mental constitution of the parents, there can be no doubt that this proportion would be much larger. Neurasthenia tends so markedly to run in families that we are led to suppose the heredity to be dominant. Nevertheless we may presume that a number of different heredity factors contribute to the causation of neurasthenia. We incline, therefore, to a hypothesis of polymeric causation, without excluding the possibility that there may also be isolated heredity factors, each of which is competent unaided to produce neurasthenia.

There are close relationships between neurasthenia and hysteria. We often encounter the symptoms of the two states in combination ("hysteroneurasthenia"). We can account for the association on the hypothesis that the neurasthenic sense of an incapacity to meet the demands of life predisposes to a hysterical flight into illness. Among neurasthenics under medical treatment there are many more men than women. The higher claims which life is apt to make upon the mental faculties of males have the effect of making an abnormal susceptibility to fatigue more frequently obvious in them than in females. On the other hand, the hysterical flight into illness comports less with the character and the social position of the male. It is possible, then, that identical heredity factors may induce neurasthenia in the male, and hysteria in the female.

The term *obsessional neurosis* is used to denote psychopathic conditions in which certain unfounded and obsessional ideas force themselves into the patient's mind and maintain themselves there in defiance of his better judgment. For instance, he may be haunted by the idea of committing a crime. According to Kehrer,[1] the predisposition to obsessions is akin to the schizoid temperament without being identical therewith. In a third of his patients who were subject to obsessions, Kraepelin found that the same predisposition was present in parents and children. J. Piltz,[2] Stöcker,[3] and F. Meggendorfer[4] have reported on families in which the taint could be traced for several generations. Kehrer writes: "From a study of the purest instances of the obsessional neurosis, such as Piltz's cases, in which the heredity could be traced for three generations, we learn that the heredity is markedly dominant." I think, however, that this supposition cannot be generalised without further enquiry.

Closely allied to the obsessional neurosis is the *anxiety neurosis*. Those affected with this latter, suffer from unfounded but irresistible anxiety in respect of certain things or processes. For instance, they may dread crossing an open space (agoraphobia), or they may dread encountering other people, or they may dread being shut up in a confined space like a railway compartment (claustrophobia). These obsessional anxieties are certainly in great measure the outcome of hereditary predisposition, and they are perhaps biologically associated with the obsessional neurosis.

Another kind of psychopathy manifests itself in the form of a *morbid excitability*, often disclosing itself in violent fits of passion.

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[1] F. Kehrer and E. Kretschmer, Die Veranlagung zu seelischen Störungen, Springer, Berlin, 1924.

[2] Ueber homologe Heredität bei Zwangsvorstellungen, "Zeitschr. f. d. ges. Neur.", vol. XLIII, 1918, p. 134.

[3] Ueber Genese und Klinische Stellung der Zwangsvorstellungen, "Zeitschr. f. d. ges. Neur.", vol. XXIII, 1914, p. 121.

[4] Ueber spezifische Vererbung einer Angst- und Zwangsneurose, "Zentralb. f. d. ges. Neurologie und Psychiatrie", vol. XXX, 1922, p. 221.

[5] Familienforschung und Vererbungslehre, second edition, Barth, Leipzig, 1922.

R. Sommer[5] has drawn attention to the way in which this disposition runs in families. K. Schneider[1] distinguishes a special group of psychopaths as "explosive psychopaths". Kehrer regards the so-called "affective epilepsy" as a manifestation of this kind of mental hyperexcitability.

For many persons, occasional outbursts of excitement seem to be a physiological need, and the extent to which this impulse to excitement prevails varies much from one family to another. A certain amount of it may be considered normal. It finds expression in popular festivals, sport, war, and revolution.

Schneider further distinguishes a group of *will-less psychopaths*, who by Kraepelin are termed "the unstable". Such persons are weak of character, easily led now in one direction and now in another, straws in the wind. Sommer points out that a readiness to be influenced is a striking character-trait in many families. This trend seems to be related to the hysterical temperament.

In addition, Schneider separates a group of psychopaths who manifest a *morbid need for self-assertion.* It is, however, difficult to draw a clear distinction between these and hysterics. I myself, in 1921, insisted that the "need for self-assertion" was one of the motive forces in persons of the hysterical temperament.

The *unfeeling psychopaths* are characterised by a lack of sympathy. Such a lack of sympathy readily leads to crime, so that this character-trend is more of a danger to society than to the individual. E. Bleuler[2] found that all these morbidly unsympathetic persons had the same sort of hereditary taint.

The psychopathies we have just been considering may be classed together as entailing a hereditary predisposition to crime, about which much has been written.

The term "moral insanity" has been employed by many psychiatrists, and has even passed into general use. This term, however, is unsuitable for the delimitation of a particular kind of insanity or psychopathy, for the idea of the "moral" is not a biological one.

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[1] Die psychopathischen Persönlichkeiten, contributed to Aschaffenburg's Handbuch der Psychiatrie, section 7, part 1, Leipzig and Vienna, 1923.

[2] Lehrbuch der Psychiatrie, Springer, Berlin.

I must not be interpreted as meaning that crime, on the one hand, and moral behaviour on the other, are not mainly determined by hereditary predisposition; what I want to emphasise is that a number of psychopathies which biologically considered are very different, are competent to induce crime. According to Schneider, the term "moral insanity" was first used by Prichard in the year 1835, to distinguish morbid disorders of the emotional life and of the character from "intellectual insanity".[1]

Schizophrenes occasionally perform acts of cold-blooded cruelty, or commit purposeless deeds of violence. To a lesser degree, the same is true of schizoid psychopaths. Paranoid psychopaths are not infrequently led to break the law by political or religious considerations. When paranoiacs commit crimes, it is more often in order to defend themselves against imaginary persecution. Epileptics are prone to commit acts of violence. Hysterics with a craving for self-assertion form a considerable proportion of swindlers and sharpers. Feeble-minded persons are very apt to commit crimes, being unable to foresee the consequences of their actions. Unfeelingness and anomalies of the impulsive life are exceptionally prone to lead to crime when they are associated with feeble-mindedness. In a study of 470 male Jews who had come into conflict with the law, J. H. Williams[2] found that 30% of them were markedly feeble-minded, and that the residue had obviously less intelligence than other youths of the same age. He considers that a lack of insight or judgment had, in these delinquents, been the main cause of crime. A large proportion of all crimes are committed by alcoholics, and inasmuch as any morbid mental disposition can help to produce alcoholism, hereditary predisposition plays its part here. Cycloid psychopaths of a hyperthymic type are apt to commit crimes from heedlessness. By no means rare among criminals are the unstable psychopaths—persons who, without being really malevolent, incline always to follow their first impulses. They will steal whatever seems to them desirable; they have no sense of truth; and they yield to their sexual inclinations regardless of consequences. Such persons are little if at all educable. Contrasted with these is the group of the unfeeling psychopaths, the "born criminals"; in whom there is no other mental disorder than an extreme lack of sympathy and sensibility, so that their lives are usually passed in an unceasing round of crime and punishment. I need hardly say that this grave mental abnormality (which, happily, is by no means common) is mainly the outcome of hereditary predisposition.

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[1] James Cowles Prichard, A Treatise on Insanity, London, 1835.

[2] The Intelligence of the Delinquent Boy, "Journal of Delinquency", Monograph no. 1, Whittier (Col.), 1919.

Gruhle, Lund, and others, have studied the morbid heredity of young persons who have run off the rails. In the sequel to the present work, this matter will be considered in connexion with the problem of social selection. There, too, an account will be given of the various extensive families of criminals and vagabonds which have been carefully studied, such as the Juke family, the Zero family, etc. Here I shall be content to refer to the investigations of C. Rath,[1] who was able in a number of families to trace a predisposition to crime through several generations. It is noteworthy that in these families criminal acts were recorded on the part of 296 males and on the part of only 44 females. Obviously members of the more active male sex are more apt to commit crimes. The predisposition, however, is transmitted just as much through women as through men.

We are indebted to the psychiatrist G. Aschaffenburg[2] for a comprehensive discussion of the biological causes of crime. K. Birnbaum[3] has given a detailed account of the psychopathic criminal.

There is just as little reason for distinguishing a special type of "degenerative insanity" as a special type of "moral insanity". For example, it does not seem obvious why schizophrenia or epilepsy should not be included within this concept of degeneration. The reasons already considered (see above, p. 312) against the notion of a "status degenerativus" and against "heredodegeneration" as biological unities, apply with equal force against the concept of a "degenerative insanity".

On the other hand, the "signs of degeneration" or "stigmata of degeneration" have special importance as concerns the domain of psychiatry. The insanities and the psychopathies usually manifest themselves in the bodily appearance as well as in the behaviour, and this not only in signs which can be regarded as consequences of the mental disturbance or anomaly (facial expression, etc.), but also—and this is especially noteworthy—to some extent in the bodily structure. Of course it is not possible from an individual's appearance to deduce in every case the nature of the mental disorder from which he suffers; but if the various insane persons in an asylum were to be arranged in groups according to the nature of their mental disorder, no one with any psychiatric experience could doubt which was the group of the schizophrenes, that of the manic-melancholics, that of the epileptics, and that of the idiots. A competent observer can in daily life recognise a good many psychopaths simply by their outward aspect (in a railway train, for instance) even when they do not wear their hair long or deliberately attempt to make much of themselves by some other peculiarity of dress or demeanour. We may distinguish between direct and indirect signs of degeneration. One of the direct signs is microcephaly, which is an unmistakable indication of feeble-mindedness. Direct signs of degeneration caused by the same heredity factors as those which are responsible for the morbid mental constitution, make themselves conspicuous in a comparatively large number of cases. According to E. Rüdin,[1] it is "a fact well known to every expert" that there is a correlation between such mental defects as imbecility and idiocy, on the one hand, and, on the other, developmental anomalies of the eye, whose number and severity increase in striking correlation with increase in the severity of the mental defect (T. Gelpke).[2] In some of the instances, certainly, this connexion may be the outcome of external noxious influences which, in the early period of embryonic life, have at one and the same time impaired the development of the central nervous system and the closely connected development of the eye. Indirect signs of degeneration are those whose connexion with mental defect depends upon an elective accumulation of various heredity factors in the same hereditary equipment. Persons with congenital mutilation or malformation will usually mate with persons similarly affected or with persons who are mentally below par, for those who are sound in mind and body will not accept them as life partners. Thus when we find in any individual that there is an accumulation of bodily defects which do not derive from the same heredity factors as mental defects, we have some ground for inferring that mental defects will also exist, though naturally not nearly as strong a ground as if we had to do with a direct connexion. For the aforesaid reason (on account of the difficulty a bodily or mental defective experiences in finding a non-defective mate), there is likewise a correlation between various psychopathic taints. Hence mental anomalies can to some extent become "signs of degeneration" as regards other mental anomalies. In the sequel to the present work, when discussing social selection, we shall speak in fuller detail of this heaping-up of mental defects in certain kinships and certain strata of the population.

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[1] Ueber die Vererbung von Dispositionen zum Verbrechen, Stuttgart, 1914.

[2] Das Verbrechen und seine Bakämpfung, third edition, Heidelberg, 1923.

[3] Die psychopathischen Verbrecher, second edition, Thieme, Leipzig, 1926.

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[1] Ueber Vererbung geistiger Störungen, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. LXXXI, 1923, nos. 3 and 4.

[2] Ueber die Beziehungen des Sehorgans zum jugendlichen Schwachsinn, Marhold, Halle, 1904.

Especially important is a correlation between mental and bodily constitution discovered by E. Kretschmer.[1] By numerous measurements he has been able to prove that among the insane of the schizophrene group the slender ("leptosome") and the athletic habit of body predominate, whereas the patients comprising the manic-melancholic group are as a rule of the stocky ("pyknic") constitutional type. Furthermore, the schizophrenes have on the average smaller heads, narrower faces, more prominent noses, and smaller lower jaws than the manic-melancholic. These results have been confirmed by various other investigators, extremely numerous measurements having been made by K. O. Henckel.[2] Kretschmer also tells us that the various groups of psychopathies respectively related to the before-mentioned main groups of the insanities are likewise correlated with the corresponding forms of bodily constitution. Finally, Kretschmer believes that he has been able to trace like connexions in the domain of normal differences of character. He classes the schizoid psychopathies, together with the analogous character-types which are not to be regarded as morbid, in the group of the schizothymic characters; and the cycloid psychopathies, together with the corresponding non-morbid characters, in the group of the cyclothymic characters. He has, however, himself insisted that human beings cannot be classed simply and without qualification in these two groups. Nor, of course, have we definitively pigeon-holed any one by character and temperament when we have ranged him in one or other of these groups. Just as little as all colours can be graded in series between black and white, just so little can the same sort of thing be done in the domain of the mental constitution.[1]

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[1] Körperbau und Charakter, third edition, Springer, Berlin, 1925; English translation by W. J. H. Sprott, Physique and Character, Kegan Paul, London.

[2] Studien über den konstitutionellen Habitus der Schizophrenen und Manisch-Depressiven, "Zeitschrift für Konstitutionslehre", vol. XI, 1925, no. 6.

We may presume that the above-described relationships between bodily structure and mental peculiarities are in great measure determined by the individual peculiarities of the internal secretions. In this connexion I may refer to the fact that the differences between the sexes also conform to the Kretschmerian typology. The males of our species incline as regards body to be leptosome or athletic, and in mind to be of schizothymic temperament; whereas women incline in habit of body to be pyknic, and in mind to be of cyclothymic temperament, and, moreover, they are more liable than men to manic-melancholic disorders. To some extent we can apply the same classification to the different phases of life. Young children incline to a pyknic bodily type and to a cyclothymic mental constitution. Towards puberty, the body takes on a more leptosome form; conformably therewith, these developmental years are especially characterised by schizothyme mental trends; and we note that the outbreak of schizophrene psychoses tends especially to occur at this age. Later, in middle life, the habit of body once more takes a pyknic turn, and in conformity therewith the mind inclines to assume a cyclothymic constitution. In the bodily and mental peculiarities of the great races of mankind we note similar group distinctions—a matter to be considered later.

Anomalies of the sexual impulse must be classed among the psychopathies. A morbid strength of the impulse towards coitus manifestly tends to run in families; and so, on the other hand, does abnormal weakness or complete lack of this impulse. The most important anomaly of the reproductive impulse is what is known as *homosexuality*, in which an individual's sexual desires are directed toward persons of the same sex. Some homosexuals are certainly to be regarded as sexual intermediate types (see above, p. 299),[2] for in homosexuals more often than in heterosexuals (persons whose sexual desire is directed towards members of the opposite sex) do we note bodily traits characteristic of the other sex.

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[1] W. Mayer-Gross, Grundsätzliches zur psychiatrischen Konstitutions- und Erblichkeitsforschung, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. C, 1926, nos. 2 and 3.

[2] R. Goldschmidt, Die biologischen Grundlagen der konträren Sexualität und des Hermaphroditismus beim Menschen, "Archiv für Rassenbiologie", vol. XII, 1916, no. 1.

But in a great many, nay in the majority of homosexuals, we are especially struck by the contrast between the bodily equipment and the direction of the sexual impulse. To some extent homosexuality may, it would seem, be originated or strengthened and confirmed by seduction, example, and habit, especially in those who are psychopathically suggestible or constitutionally unstable. On the other hand, we sometimes note a confirmed homosexual trend in persons who in other respects manifest no signs whatever of psychopathy.[1] While it is true that this disorder of the sexual impulse has, so far, seldom been found to run in families,[2] the lack of evidence in this respect may be due to the difficulty of ascertaining the existence of a predisposition which is commonly regarded as vicious. As the psychiatrist F. Kehrer[3] writes: "At any rate there can be no doubt, nowadays, as to the existence of an isolated perversion of the sexual impulse, a perversion which is exclusively determined by the germinal equipment, usually manifesting itself at puberty, persisting throughout life, and more or less independent of the psychogenic influences of the environment." Homosexuality, however, cannot be regarded as a normal or quasi-normal variant, as its organised defenders are wont to declare. It is unmistakably morbid, being markedly antagonistic to race preservation.

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[1] A. Kronfeld, Sexualpsychopathologie, contributed to Aschaffenburg's Handbuch der Psychiatrie, Leipzig and Vienna, 1923.

[2] J. Piltz, Homologe Vererbung der Homosexualität, "Zentralblatt f. d. gesamte Neurologie und Psychologie", vol. XXVI, 1921, p. 76.

[3] Ueber Wesen und Ursachen der Homosexualität, "Deutsche Medizinische Wochenschrift", 1924, no. 19.

CHAPTER TEN

THE ORIGINATION OF HEREDITARY MORBID PREDISPOSITIONS

All morbid heredity factors must, of course, have originated at some time or another. It is true that we can trace back certain morbific heredity factors through numerous generations; for instance, we can trace back night-blindness in one genealogical tree for more than three centuries; and recessive heredity factors can exist in a population for many generations, ambushed as it were, before they manifest themselves for the first time; but, whether dominant or recessive, these factors must have originated at some time or other. It is obvious that in the hereditary equipment of the first human beings there cannot yet have been present all the morbific heredity factors with which our present populations are tainted. Inasmuch as biologically there is no essential distinction between morbific and normal heredity factors, the morbific heredity factors must have originated in the same sort of way as the heredity factors that are termed normal. Since we have to represent to ourselves the hereditary equipment as of a chemical-physical nature, it must be subject to alteration by chemical-physical influences; and since the individual heredity factors, owing to their molecular structures, cannot be connected by a continuous series of transitions, changes in them, when these occur, must occur "by jumps", through the loss, addition, or rearrangement of molecules or groups of molecules. We term "idiokinetic" influences those chemical or physical influences which produce changes in the hereditary equipment; and the causative process whereby such variations in the hereditary equipment (idio-variations) are brought about is spoken of as "*idiokinesis*".[1]

This word "idiokinesis" is not identical in meaning with the term "blastophthoria" (injury to the germ) introduced by Forel, for this latter denoted non-hereditary as well as hereditary changes, and was limited to alterations for the worse. Following Siemens, we use the term *parakinesis* to denote the process which gives rise to non-hereditary changes, whether in the completed living being or in the germ cells; and we therefore speak of parakinetic influences in contradistinction to idiokinetic influences.

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[1] *τό ἴδιον*, private concerns, essential nature; *κινεῖν*, to set in motion, to change.

As regards matters of detail, the determinants of idiokinesis are still very imperfectly known, but this does not affect the theoretical soundness of the concept.

In European biological literature, some of the results obtained by the American zoologist Tower have been widely quoted as proofs of the experimental production of inheritable changes. Tower believed that by exposing the potato beetle (Leptinotarsa) to abnormal temperatures he had produced hereditarily transmissible changes in that insect. In America, Tower's publications have been regarded sceptically; and it would, indeed, seem that his results imply nothing more than the mendelising out of certain biotypes already present in the hereditary equipment. At the Dahlem Biological Institute, V. Jollos[1] believed that he had been able to produce in unicellar animal organisms, by the effect of arsenious acid (and also by abnormal temperatures), not only a number of permanent modifications, but also a few genuine mutations. In some cases the "permanent" modifications did, indeed, persist through many asexual generations, but after one (or in exceptional instances after a few) conjugations the organisms returned to the original type. The mutations, however, persisted after repeated conjugations.

In our population, *alcohol* would seem to play an important part among the causes of morbid hereditary taints. In the offspring of alcoholics various kinds of mental disorders and feeble-mindedness are found in much above average frequency; and in some of the cases, at least, these changes seem to be hereditary. About one third of all epileptics, imbeciles, and idiots are the progeny of hard drinkers. Of course in many of these cases it may well be that the tendency to drunkenness in the parent (usually the father) was a manifestation of the same heredity factor which took effect in the children as epilepsy or imbecility. Among the parents of schizophrenes, too, we find an unusually large number of alcoholics, for it would appear that the schizoid temperament predisposes to alcoholism. In individual cases it is impossible to decide whether alcoholism is the cause or the effect of a low-grade mentality. In my opinion, however, there can be no doubt that the abuse of alcohol has idiokinetic effects, and that in practice these effects are considerable.

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[1] Experimentelle Protistenstudien, "Archiv für Entwicklungsmechanik", 1921; republished in book form by Fischer of Jena.

The persistent abuse of alcohol leads in the end to a destruction of the testicular cells out of which the spermatozoids are formed. E. Bertholet[1] found in postmortem examination of the bodies of 163 alcoholics that in 140 of them (= 86 ± 3 %) the germinal epithelium of the testicles had atrophied, whereas in 100 bodies of non-alcoholics such atrophy was found only in 29 (= 29 ± 5 %). In 23 drinkers who died a violent death, in 16 (= 69 ± 10 %) the germinal epithelium was atrophied, whereas in 21 non-drinkers who had died a violent death there was no such atrophy in any of the cases. This explains why hardened drinkers in the long run usually become sterile. In alcohol, we have a poison which is capable of destroying the germinal epithelium, and it would be very remarkable if this poison, acting with less intensity, did not sometimes produce changes in the hereditary equipment contained in the germinal epithelium.

Numerous attempts have been made to prove by experiments on animals that alcohol has a deleterious influence on the hereditary equipment. Noteworthy, in this connexion, are the results obtained by the American anatomist C. R. Stockard[2] at the medical school of Cornell University. Taking guineapigs which had previously brought forth normal young, Stockard exposed them daily (Sundays excepted!) for months to the fumes of alcohol. The animals did not become obviously ill, but were never quite sober, their condition thus corresponding to that of a good many "moderate" drinkers. When such alcoholised guineapigs were mated, in a good many instances they proved sterile; there were also numerous stillbirths; and among the few young born alive, a large majority died prematurely—a rare occurrence in the offspring of non-alcoholised guineapigs used in control experiments. Even when only the mother or only the father was alcoholised, various states of enfeeblement and various malformations appeared in the young; and when such degenerate animals were paired, the morbid taint appeared in the second, and even in the third or fourth generation.

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[1] Action de l'alcoolisme chronique sur les organes de l'homme et sur les glandes réproductrices en particulier, Frankfurter, Lausanne, 1913.

[2] C. R. Stockard and G. Papanicolaou, Further Studies on the Modification of the Germ Cells in Mammals; the Effect of Alcohol on treated Guineapigs and their Descendants, "Journal of Experimental Zoology", 1918. See also the summarised report in A. Bluhm's Alkohol und Nachkommenschaft, "Zeitschrift für induktive Abstammungs- und Vererbungslehre", 1922.

The investigations of R. Pearl[1] on fowls seem to me devoid of evidential value, for he worked with material in which already before the parent birds had been poisoned with alcohol 42 % of the embryos died before the eggs were laid. Equally without evidential value seem to me F. Bilski's[2] experiments on frogs, for he was working upon much too small a number of animals, and furthermore the animals in his control breedings had far too high a mortality (as much as 90 %). E. Rost and G. Wolf,[3] administering alcohol in doses up to 10 cc. daily to rabbits for 2½ years, were unable to detect any changes in the organs of these rabbits or any injury to their offspring. But the number of animals used in their experiments was so small (only 7 females in all!) that we are not entitled to infer that no changes in the heredity factors can be induced by alcohol—all the more seeing that not in a single instance were the offspring bred beyond the F_2 generation.

The Belgrade psychologist A. Kostitsch[4] believes that in white rats he has been able to produce recognisable injury to the spermatozoids by the administration of alcohol. For a long period the experimental animals received a daily dose of about 1·4 c.c. of alcohol. The result was that in the course of time the spermatozoid-forming cells of the testicles perished, whereas the interstitial cells remained uninjured. Although the power of copulation was not markedly affected, sterility ultimately ensued. Soon after he had begun to administer the alcohol, Kostitsch was able to detect all kinds of disturbances in the process of nuclear division, noting in especial that in the division the two daughter cells received different quantities of nuclear substance. Only in a small proportion of the cells was development arrested; whereas irregularities in the developmental process were manifest in the majority.

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[1] The Experimental Modification of Germ Cells, "Journal of Experimental Zoology", 1917.

[2] Ueber Blastophthorie durch Alkohol, "Archiv für Entwicklungsmechanik", 1921.

[3] Zur Frage der Beeinflussung der Nachkommenschaft durch Alkohol im Tierversuch, "Archiv für Hygiene", vol. XCV, 1925, nos. 3 and 4.

[4] Action de l'alcool sur les cellules séminales, "Internationale Zeitschrift gegen den Alkoholismus", 1922, no. 2.

E. C. MacDowell and E. M. Vicari[1] report that in the grandchildren of white rats which had been alcoholised for a long time, the capacity for learning and the memory were impaired.

We must certainly admit that the experiments hitherto made concerning the production of inheritable modifications by alcohol are far from satisfying. Indeed, the difficulties in the conduct of trustworthy experiments are almost insuperable. One of the chief of these difficulties lies in the fact that in the animals that have been used for such experiments (rabbits, guineapigs, rats, white mice), definite characters or hereditary traits can scarcely be verified by measurement. For this reason the usefulness of the white mouse is greatly restricted, although in other respects this animal is most suitable on account of its cheapness. Experiments with other animals entail great expenditure upon attendance and food, seeing that, for a single series of experiments, if trustworthy results are to be secured, hundreds of animals must be bred and kept under observation. Nevertheless, in view of the importance of the question, it is eminently desirable that trustworthy experiments on animals should be conducted on a large scale. I may remark in this connexion that Alfred Ploetz, the founder of racial hygiene in Germany, has for some years been engaged in such investigations upon an extended scale. We shall, however, have to wait for a good while yet before decisive results can be expected.

From idiokinetic damage to the offspring, we must be careful to distinguish a purely parakinetic damage, in which the hereditary equipment is unaffected. Thus the frequent occurrence of miscarriages in the families of alcoholics must, no doubt, be partly attributed to direct poisoning of the fruit through abuse of alcohol on the part of the mother; and the extraordinarily high child mortality in the families of alcoholics must in great measure depend upon the disorder of domestic life in such households, though certainly it is in part due to impaired hereditary equipment. When the offspring suffer from general debility because the father was a drunkard, we can hardly suppose that the injury has been merely parakinetic and must assume that generally speaking it has been idiokinetic. We can hardly suppose that alcoholism in the father can influence the offspring parakinetically, seeing that the spermatozoid, having a millionfold smaller mass than the egg cell, cannot bring with it into the fertilized ovum a quantity of alcohol capable of having an appreciable effect. Parakinetic effects capable of being subsequently counteracted must therefore be derived exclusively or almost exclusively from the mother. In conformity with this expectation is Bilski's observation that alcohol has a more destructive influence upon the ovum than upon the spermatozoid; and also Agnes Bluhm's observation that the alcoholisation of female white mice has a disastrous effect upon their fertility, whereas the alcoholisation of male white mice reduces their fertility very little. On the other hand, in the light of these observations and considerations, very great importance must be attached, as proof of a genuinely idiokinetic influence, to the experiments of Stockard and of Bluhm in which only the paternal animals were alcoholised. In Agnes Bluhm's experiments, of 254 mice born alive and procreated by alcoholised fathers, 161 died during the first two months of life, this being a mortality of 64 ± 3 %, whereas among 839 control animals only 391 died, that is to say 47 ± 1·7 %.

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[1] Alcoholism and the Behaviour of White Rats: I, The Influence of Alcoholic Grandparents upon Maze-Behaviour, "Journal of Experimental Zoology", vol. XXXIII, 1921, p. 209.

Stockard's results were obviously no mere "permanent modifications", no mere "induction" as the Lamarckians phrase it; for the inherited changes persisted through sexual reproductions for several generations. In multicellular animals, the numerous cell divisions that occur during individual development correspond to the asexual generation of unicellular animals. Consequently, in multicellular animals, we should expect "permanent modifications" to last as a rule only for an individual lifetime,[1] and they would certainly not last for four generations as did the hereditary modifications produced by Stockard. In conformity with this we find that a sanation of the stock in which Stockard had artificially produced morbid modifications only occurred in proportion to the extent to which there ensued a crossing with sound stocks and a natural selection through the dying-out of diseased individuals. Stockard worked on the large scale, breeding many hundreds of individuals, and his results cannot be attributed to chance.

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[1] We must, for instance, regard the normal difference between the various tissues as "permanent modifications", seeing that idiotypically all the cells of the body are alike.

In Germany before the war, the consumption of pure alcohol per head of population was about 8 litres, this being the amount of alcohol contained in approximately 200 litres of beer. Since the greater part of this amount is drunk by males at an age when they are capable of procreating, we may assume that a German male during the years of active sexual life consumes annually at least 25 litres of pure alcohol, this corresponding to a daily consumption of at least 2 litres of beer. In the towns the abuse of alcohol was, of course, even greater. Thus in Munich, during the year 1907, the consumption of beer per head of population amounted to 287 litres. During the war and the post-war years, the consumption was much smaller; but since 1924 a great increase has been noted, and to-day nearly as much beer is drunk as before the war.

It is a moot question whether transient alcoholic intoxication can result in the procreation of degenerate offspring. Some have actually supposed that pairing while in a state of intoxication may be disastrous because the fugitive intellectual disorder of an intoxicated person may be transmitted to the offspring. I need hardly say that nothing of the kind is possible. It is, however, conceivable that spermatozoids stored in the seminal vesicles may be injured by alcohol circulating through the body, and that such a spermatozoid, when it has so to say slept off its debauch, might in conjugation with a healthy ovum lead to the formation of a child with hereditary injuries. D. Bezzola,[1] studying the date of birth of 8186 imbeciles and idiots, found that a comparatively large proportion of these must have been engendered during carnival and at the time of vintage. E. H. Müller[2] has noted a similar deviation from the normal distribution of birth dates in the case of 847 epileptics. But these deviations from the normal distribution—deviations which are by no means extensive—might also be interpreted on the hypothesis that individuals who are below par mentally are more likely than others to propagate their kind at such seasons. The experiments of L. J. Cole and C. L. Davis[3] have established beyond question that spermatozoids in the body can be influenced by alcohol. When a doe rabbit was covered in rapid succession by two bucks of different races, one of which had been given a dose of alcohol, the procreated young were the offspring of the male which had not been alcoholised. But if an alcoholised male was alone permitted to copulate, it was capable of procreating young.

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[1] Statistische Untersuchungen über die Rolle des Alkohols bei der Entstehung des originären Schwachsinns, "Internat. Monatsschr. f. Bekämpfung des Alkoholismus", vol. XI, 1902, p. 171.

[2] Einige Beziehungen des Alkolismus zur Aetiologie der Epilepsie, "Monatsschrift für Psychiatrie und Neurologie", vol. XXVIII, 1910, p. 1.

[3] The Effect of Alcohol on the Male Germ Cells studied by Means of Double Matings, "Science", 1914.

Nevertheless we have to note that in the experiments of O. Hertwig, frog spermatozoids immersed for hours in a 5 % solution of alcohol retained the power to fertilise the ovum, and that ova thus fertilised developed into apparently normal embryos. Bilski reports that Ivanoff was actually able to induce artificial fertilisation with the sperm of rats, sheep, dogs, rabbits, and guineapigs which had been treated with 7 % alcohol; and that the offspring in these cases were normal. It is, no doubt, possible that idiokinesis can hardly be brought about by alcohol at any other time than during the maturation (ripening) of the reproductive cells, and Kostitsch's data favour this view.

It may be that an indirect injury to the hereditary equipment may result from alcoholism, through the working of toxic products of metabolism which may be formed in the alcoholised body. The abuse of alcohol may cause grave injury to almost all the organs (the stomach, the bowel, the liver, the kidney, the heart, and the nervous system). Now, one of the normal functions of the liver is a detoxication of the body, and therefore when the liver is damaged such toxic products of tissue change as cholin may work mischief that would not ensue if the liver were healthy.

In addition to alcohol, many other poisons have to be taken into consideration as possible causes of idiokinesis, especially those poisons which are used in industrial processes. Among such poisons may be mentioned *lead*, *mercury*, *phosphorus*, *nicotin*, *carbon disulphide*, *benzol*, *anilin*, and kindred substances.

It has been reported that the offspring of lead workers are often sickly when the mother was not exposed to lead poisoning. O. Rennert,[1] for instance, found that in the families of 11 potters among 79 children 56 were sickly. Convulsions and a peculiar enlargement of the head are common among the children of lead workers. Experiments in lead poisoning have been made on animals. Professor Cole of the University of Wisconsin had doe rabbits covered by a male which had been treated with lead and immediately afterwards by another male which had not been treated with lead and which belonged to a different race. The upshot was that the offspring of the male treated with lead were weaker and sicklier than those in the same litter procreated by the untreated male. When roosters were poisoned with lead, there was a much higher than average mortality among the embryos in the eggs of the hens fertilised by them; and there was an exceptionally high mortality among the chickens likewise.[1] C. V. Weller[2] found that the offspring of lead-poisoned male guineapigs were lighter by one fifth than control animals, showed a retardation of development, and had a higher mortality. Unfortunately in these experiments, as in those of Cole, only the offspring of the first generation were observed.

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[1] Ueber eine hereditäre Form chronischer Bleivergiftung, "Archiv für Gynäkologie", vol. XVIII, 1881, p. 109.

As regards nicotin, comprehensive experiments on animals are urgently desirable. The frequency of miscarriages among female tobacco workers, and the no less notable frequency of impotence in men who are heavy smokers, lead me to regard it as very probable that tobacco will be found in practice to be one of the most important among the noxious influences capable of producing diokinesis.

Idiokinetic influences are also to be expected in the case of a number of substances used in medicine, especially the protozoa-killing poisons like *quinine*, *mercury*, *iodine*, and *arsenic*. In experiments on animals we find that comparatively small doses of free iodine (doses so small as to have no other noteworthy effect) can produce transient sterility; and that larger doses can produce permanent sterility.[3] All the aforesaid poisons are used to destroy animal micro-organisms when, as exciters of disease, they have invaded the human body; for instance, the organisms that give rise to malaria and to syphilis respectively: but since these same poisons can destroy the fruit in the mother's womb, we know that they can also prove deadly to human cells. It is, however, absurd to suppose that by such a poison a reproductive cell will either be slain outright or else left entirely uninjured. Between these extremes there lies the possibility of a more or less extensive injury to a reproductive cell and to its hereditary equipment.

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[1] L. J. Cole and L. F. Bachhuber, The Effect of Lead on the Germ Cells of the Male Rabbit and Fowl, as indicated by their Progeny, "Proceedings of the Society of Experimental Biology", vol. XII, 1914, p. 24.

[2] The Blastophthoric Effect of Chronic Lead Poisoning, "Journal of Medical Research", vol. XXXIII, 1915, p. 271.

[3] Adler, Ueber die Jodschädigungen der Hoden, "Archiv für experiment. Pathologie und Pharmakologie", vol. LXXV, no. 5; Loeb and Zoeppritz, Die Beeinflussung der Fortpflanzungsfähigkeit durch Jod, "Deutsche Medizinische Wochenschrift", 1914.

If we take a comprehensive view of the poisons which more than most are suspected of exerting injurious idiokinetic influences, we find that they are characterised by certain similarities in their other effects. All of them when their working is long continued lead to a profound bodily decay (cachexia, marasmus), so that we speak of alcoholic cachexia, lead cachexia, mercurial cachexia, iodine cachexia, arsenical cachexia. Manifestly, here, we have to do with an injury to the idioplasm of the body cells; for unless the idioplasm is injured all disorders of the cells can be made good. Furthermore, every one of the before-mentioned poisons has, when its working is continued for a long time, the capacity for producing severe nervous disorders, usually in the form of multiple neuritis, with tremor and disorders of vision; more especially the optic nerves are liable to be affected by these substances; alcohol, tobacco, arsenic, lead, mercury, carbon disulphide, and quinine not infrequently cause grave impairment of vision and even complete blindness. Speaking generally, the sensibility of the central nervous system to poisons is closely akin to that of the reproductive cells. In the case of all narcotics, therefore, we have reason to suspect that idiokinetic effects will result from long-continued exposure to their influence. Thirdly, common to all these poisons is the power of destroying the fruit in the mother's womb and thus giving rise to abortion.

Idiokinetic effects have been clearly demonstrated in the case of *Röntgen rays*, and also in the case of the radiations of radioactive substances, whose influence is akin to that of X-rays.

Oskar Hertwig,[1] exposing the spermatozoids and the ova of amphibia to the radiations of radioactive substances, found that even in the experiments in which only the spermatozoids were thus exposed, the individuals proceeding from the development of normal ova which had been fertilised by such spermatozoids exhibited various malformations and states of enfeeblement. He obtained similar results through the action of certain chemicals (methylene blue, chloral hydrate, quinine) upon mature spermatozoids. In this connexion he writes: "These experiments with radioactive substances and with substances whose action is chemical have given irrefutable proof that in this way the idioplasm of the reproductive cells can be permanently modified." Hertwig goes on to infer (rightly, in my opinion) "that within the reproductive glands the ova and the spermatozoids can be modified by the influence of radium or of X-rays just as they can be modified by these when isolated for experimental purposes".

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[1] Die Radiumkrankheit, "Archiv für mikroscopische Anatomie", 1911; Das Werden der Organismen, second edition, Fischer, Jena, 1918.

By an unexceptionable and systematic series of experiments, the American zoologist H. J. Muller (formerly a collaborator of T. H. Morgan, and now professor at the University of Texas) has shown that in the fruit-fly Drosophila numerous hereditary modifications can be deliberately induced by Röntgen rays.[1] By exposing both male and female fruit-flies to the rays, Muller was able to produce a great many mutations, which persisted for three, four, or more generations. The inheritance of these mutations took place in accordance with the mendelian law, just like the inheritance of those mutations which are observed to arise "spontaneously" as an effect of the ordinary influences of life. During a few months of experimental work, Muller was able to produce hundreds of mutations, almost as many as the spontaneous mutations that had been observed during the whole previous period of genetic research. The frequency of mutations in the breeds exposed to the radiations was about 150 times as great as the frequency in the control breeds that were not exposed to the rays. Most of the mutations thus induced were sex-linked, and must therefore be supposed to have been localised in the X-chromosome—the simplest and most obvious explanation of this being that sex-linked heredity factors are the ones most accessible to genetic analysis. For the rest, there was ample indication that mutations were being produced just as frequently in the other chromosomes. When the radiations were as powerful as could be borne without proving fatal, sex-linked mutations appeared in about one seventh of the offspring. Since, however, the X-chromosome constitutes only about one fourth of the haploid gene, Muller infers that in actual fact only about every other reproductive cell capable of development had undergone a hereditary modification. The necessary control experiments were made on thousands of untreated fruit-flies.

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[1] Artificial Transmutation of the Gene, "Science", vol LXVI, 1927, no. 1699.

I need hardly say that the results obtained by Muller are of epoch-making significance. The occurrence of idiokinesis and its artificial production have now been experimentally proved.

The possibility of idiokinetic injury in human beings by exposure to Röntgen rays has been contested by L. Nürnberger,[1] but upon the ground of utterly inadequate experiments and reasoning. Important, none the less, is Nürnberger's statement: "In a great X-ray establishment it is impossible, with the best precautions in the world, to avoid exposure. Sufficient evidence of this is the fact that so many of the operatives and doctors concerned in Röntgen-ray diagnosis and treatment are still sterile to-day." Of 30 married röntgenologists, 13, that is to say nearly half, had no children at all; the remaining 17 had, on the average, 1·2 children each, 21 children in all, of which 19 were presumably normal. Nor has it been satisfactorily ascertained how many of the healthy children had been procreated in the days before the father began to work with X-rays.

As regards radioactive substances, their effect is of no practical importance, since the substances in question exist in such small quantities. X-rays, on the other hand, which can be produced in unlimited quantities and are finding wider and ever wider applications for the purposes of diagnosis and treatment, may be all the more disastrous in their effects because the doctors, technicians, assistants, and nurses who deal with the rays are persons who have been selected as of above-average efficiency. Almost every practising physician is to-day exposed to the risk of injury to his germ cells by X-rays. In some places, too, most cases of tuberculosis are treated by Röntgen rays.[2]

It is probable that idiokinetic changes can most readily be brought about during the maturation of the reproductive cells, for at this time the nuclei of these cells are engaged in the complicated processes of division, and are much less efficiently protected than when they are in a state of repose.

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[1] Können Strahlenschädigungen der Keimdrüsen zur Entstehung einer kranken oder minderwertigen Nachkommenschaft führen? "Fortschritte auf dem Gebiet der Röntgenstrahlen", vol. XXVII, 1919, p. 369.

[2] Cf. F. Hilpert, Die Behandlung der Tuberculose mit Röntgenstrahlen, "Münchener Medizinische Wochenschrift", 1922, no. 10.

This hypothesis is confirmed by the fact that under the influence of iodine and kindred poisons, on the one hand, and under the influence of X-rays, on the other, the reproductive cells are most liable to be injured or destroyed during the period of maturation, and death of the embryo from the same cause is especially likely to occur in the early days of intra-uterine life, when almost all the cells are undergoing repeated divisions. We may well suppose, too, that when chemical abortifacients are administered (one of the poisons in the before-mentioned group, or perhaps aloes or juniper), and fail of their effect, they may produce idiokinetic injury in the idioplasm of the child; or they may in any case damage the germ plasm of the mother. Nor can we leave out of account the possibility of damage to the hereditary equipment by chemical substances used in order to prevent conception. All these means are more or less uncertain in their effects; and when a spermatozoid is not completely destroyed, but, though injured, succeeds in fertilising an ovum, it is conceivable that irremediable damage to the hereditary equipment may ensue.

Syphilis is often included in an enumeration of the causes of hereditary degeneration. It is certainly possible that the metabolic products which are formed in the course of this disease may sometimes have an idiokinetic effect. In the main, however, the resemblance between the effects of syphilis and those of the idiokinetic poisons may be presumed to be superficial. When diseased children are born as the offspring of syphilitic parents, it is because these children have been directly attacked by the exciting cause of syphilis while in the mother's womb. It has, indeed, often been asserted that the children of syphilitic parents are apt to be weakly and diseased even when they are free from actual syphilis. A. Peiper,[1] however, has undertaken a critical examination of these contentions (which derive from an earlier day), and has come to the conclusion that there is no satisfactory evidence of the production of idiokinesis by syphilis.

Of course this does not justify the conclusion that no such idiokinesis can occur. But even if it were found that the syphilis-free children of syphilitic parents are prone to be weakly and to lack the power of resisting disease, we have much better grounds for ascribing this to the idiokinetic effect of the indispensable poisons (compounds of mercury, iodine, and arsenic) used in the treatment of the malady. An approved way of treating syphilis is to inject intramuscularly considerable quantities of one of the less soluble salts of mercury. From the seat of injection, small quantities of mercury are continuously being poured into the circulation, thus hindering the growth of the syphilitic virus. Obviously in this way injury to the offspring can be produced just as it can be produced by the inhalation of mercurial fumes or mercurial particles in industrial occupations. As regards the arsenical compounds (salvarsan, etc.) used in the treatment of syphilis, we have less reason to expect injury to the hereditary equipment from these, for arsenic is eliminated far more rapidly.

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[1] Ist Syphilis ein Keimgift? "Medizinische Klinik", 1922, no. 12.

Speaking generally, the nature of mutations seems to depend less upon the nature of the idiokinetic influences than upon the previous constitution of the hereditary equipment. Manifestly, certain hereditary equipments are peculiarly susceptible to idiokinetic influences. E. Baur[1] and T. H. Morgan[2] in their breeding experiments found races of plants and animals which had a remarkable proneness to mutations. To use an architectural image, we may suppose that some of the stones in the edifice of the hereditary equipment were loosely built in, and could therefore be more readily removed than others. Naturally it is easier to remove such a loose stone from a building than to build in a new one. In conformity with this we find that the majority of all mutations whose appearance has hitherto been experimentally observed are recessive as against the original racial form. In Morgan's breeding experiments, during which more than 400 mutations have already been observed, only a few of them were not recessive. We must remember, in this connexion, that dominant mutations are much easier to detect than recessive ones, so that the preponderance of recessive mutations must be even greater than actually appears. It is noteworthy, too, that most of Morgan's mutants had less capacity for adaptation than had the original racial forms, and must therefore be regarded as morbid. In comparatively few only was there not plainly discernible a reduction in the likelihood of self-preservation. The same remark applies to the mutations which Baur and other investigators have observed. Besides, the morbidity will not necessarily be obvious at the first glance. Many of the mutations in Morgan's breeding experiments were only manifest in this respect, that their carriers[1] perished at some particular stage of development, or that the carrying reproductive cells were not viable (lethal factors).

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[1] Untersuchungen über das Wesen, die Entstehung und die Vererbung von Rassenunterschieden bei Antirrhinum majus, "Bibliotheca Genetica", vol. IV, Leipzig, 1924.

[2] T. H. Morgan, C. B. Bridges, and A. H. Sturtevant, The Genetics of Drosophila, Nijhoff, The Hague, 1925.

The results of such experiments in hereditary modification are strongly opposed to every kind of Lamarckism and vitalism—opposed, that is to say, to the doctrine of a direct general adaptation. The mere fact that there are morbific heredity factors which are at all capable of unlimited hereditary transmission shows that a hereditary equipment is not competent to overcome its defects by direct adaptation. The night-blind members of the Nougaret family (see above, pp. 252-3) have generation after generation made ever-renewed efforts to see in the twilight, without in this way producing any effect on the hereditary equipment. Nor do we find that mutations are generally in the direction of increased adaptation, as they ought to be were the Lamarckian and vitalist doctrines sound, and as Lamarckians and vitalists are perpetually declaring them to be. On the contrary, we find as a rule that mutations (I repeat) are in the direction of diminished adaptation. These facts of experience are readily comprehensible in the light of the converse view, the mechanist view of organisms. The various species of living creatures have for countless millenniums been so closely adapted to the conditions of their existence that—as a rule, if not invariably—a change in the hereditary equipment must involve an impairment of vital efficiency. In a living organism perfectly adapted to the conditions of existence, there could be no hereditary modification other than an unfavourable one. In the case of actual organisms, whose adaptation to the conditions of existence goes very far but is not perfect, most hereditary modifications will be unfavourable, but not necessarily all. Such is Morgan's way of looking at the matter.[2]

Since the carriers of morbific mutations of a grave kind often fail to achieve reproduction, a further hereditary transmission of the taint in these superlative cases will naturally not occur. That is the probable explanation of some of the cases in which a morbid modification known to be hereditary has appeared in one instance only in an otherwise healthy family. We have already learned, moreover, that in the case of recessive troubles isolated instances are likely to manifest themselves simply as an expression of the recessive heredity. We must also remember that mutations whose heredity is dominant will necessarily remain isolated unless reproduction is effected (see p. 407 and p. 527).

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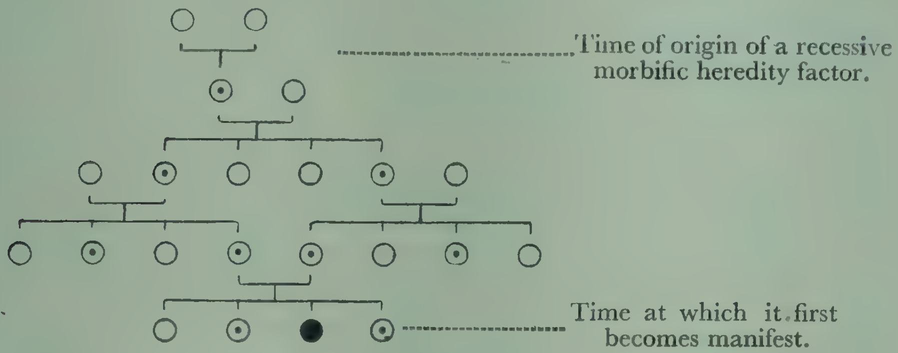
[1] A "carrier" of a hereditary character is an individual in which the character is manifest, not one in which it merely exists as a latent taint.—TRANSLATORS' NOTE.

[2] T. H. Morgan, Evolution and Genetics, 1925.

The upshot of these considerations is that the gravest disturbances of the hereditary equipment are not the ones which have the gravest significance for the life of the race. When the effects of a mutation are very serious, the reproductive cells perish, and for this reason the disorder of the hereditary equipment cannot propagate itself. Even when the injury is not quite so severe as this, the fruit often perishes in the womb, so that in these cases likewise there can be no question of a perpetuation of the morbid mutant. When the injury is somewhat less severe, the offspring often die very soon after birth from congenital lack of vitality; and when the injury is still less, the children may die before attaining the age at which reproduction becomes possible. Consequently, the injuries to the hereditary equipment which entail the most serious menace to the vital efficiency of the race are those comparatively slight ones which are so far compatible with life that their carriers can continue to exist and to propagate.

Since most of the mutations hitherto observed have been recessive, we cannot expect to find as a rule that idiokinetic damage to an individual hereditary equipment will already manifest itself in the children of the first generation. It will be most unlikely that the hereditary equipment of the other parent will be characterised by precisely the same defect, and consequently for the time being the defect will be masked. We can illustrate this by a diagram.

Let us assume that a certain heredity factor in the reproductive cell of a drinker is destroyed by alcohol. Then in the child of this drinker a corresponding recessive morbific factor will exist in a masked form (indicated by a dot within a circle). Since, in general, this child will mate with some one not harbouring the same taint, the morbific heredity factor will not manifest itself in the grandchildren. Part of these grandchildren, however, will carry the taint in a masked form. Since all the harbourers of the morbific heredity factor in the grandchildren's generation are brothers and sisters, in ordinary circumstances there will be no question of their mating one with another, and for that reason the taint will not become manifest in the great-grandchildren's generation either. But among the great-grandchildren, half of those whose parents harboured the taint will themselves harbour it. In this generation, therefore, first cousins both harbouring the taint may marry, with the result that in the next generation there may be born a child or children in whom, at last, will become manifest the disease for which the foundations were laid by the mutation in the hereditary equipment of the great-great-grandparents.



Time of origin of a recessive morbific heredity factor.

Time at which it first becomes manifest.

FIG. 156.

Diagram illustrating the first appearance of a recessive morbific heredity factor.

When, therefore, in any population a recessive taint makes itself manifest to-day for the first time, we may assume that the morbific heredity factor really came into existence more than a century ago, somewhere about the time of the Napoleonic wars. Not until now could it possibly become manifest. But since the marriage of first cousins is exceptional, we may infer that the origin of recessive taints which only become manifest to-day, may date back to the days of the Thirty Years Wars, or may have come into being at an even earlier time. In this connexion refer to Fig. 141, showing the pedigree of a case of Friedreich's disease which appeared ostensibly de novo. In the case of other recessive maladies, the conditions may naturally be supposed to be much the same. The new appearance of such troubles will be furthered by a rapid increase in the population, such as has been going on in almost all the countries of Europe since the eighteenth century. For when a population multiplies itself fourfold, let us say, within a particular time, harbourers of this newly developed recessive heredity factor will on the average have likewise multiplied themselves fourfold and there will be given increased facilities for their coming together in consanguineous marriages. But if, on the other hand, the population is not increasing, then in general newly developed recessive heredity factors will remain isolated and therefore hidden.

In experiments on animals, where pairing between brother and sister organisms can be deliberately effected, recessive morbific-heredity factors produced by injury to the hereditary equipment can become manifest already in the F_2 generation. In Muller's experiments we have ample proof of this.

Dominant mutations, of course, are already manifest in the offspring of the first generation. Among recessive mutations, sex-linked mutations occupy a peculiar position. Since in the male sex there is only one sex-chromosome, injuries to the heredity factors in the sex-chromosome can be masked in females but not in males. This, perhaps, is in part the explanation why among miscarriages males greatly preponderate in number over females—the ratio being somewhere about three to two. The mortality of boys is greater than that of girls during infancy and early childhood (see above, p. 342). On the other hand, recessive injuries to the other chromosomes are, to begin with, masked in the male sex as well.

In this connexion, reference may be made to the fact that Stockard, in his experiments upon the alcoholisation of guineapigs, found that alcoholisation of the mothers was principally dangerous to the sons. Possibly the fact that in Stockard's experiments alcoholisation of the mother was in general more detrimental to the offspring than alcoholisation of the father, is partly to be interpreted in the same sense. If the mortality of the offspring of control animals be taken as 100, then that of the offspring of alcoholised fathers is 178, and that of the offspring of alcoholised mothers 281. We have to take into account also the after-effects of injury to the protoplasm of the ova.

There is an additional reason why the effect of idiokinetic influences may differ in the two sexes. For whereas the male reproductive cells are during the period of active sexual life perpetually being formed anew out of the spermatoblasts in the testicles, substantially all the ova already exist (although immature) in the ovaries when the period of active sexual life begins, and indeed before birth—their number in the ovary of the female infant being variously estimated at from 40,000 to 100,000. Since we have reason to suppose that idiokinetic injuries chiefly arise during the processes of cell-division by which the reproductive cells come into being, in females such injuries are far less likely to occur during adult life than during the period of embryonic development. On the other hand, it may well be that the ovum, because of its greater richness in protoplasm, is more liable to idiokinetic injury than is the spermatozoid. We really have, as yet, no trustworthy information as to the distinctive ways of reaction of the respective reproductive cells towards idiokinetic influences, but it is probable that we could learn a good deal in this respect from experiments on animals.

As causes of the origination of new morbific heredity factors we must, it need hardly be said, take into consideration other influences than those of the before-mentioned poisons and other gross injuries. When animals are transferred from a free life to the conditions requisite for artificial breeding, this alone seems to be sufficient to produce morbific heredity factors in large numbers (see above, p. 173).

When, for instance, a species of butterflies is bred in captivity, so marked a degeneration sets in after a very few generations that the continuance of this particular series of experiments becomes difficult or even impossible. I have myself again and again had experience of this. The numerous mutations which Morgan and his pupils have observed in the American fruit-fly may be attributable in great measure to the unnatural conditions of the artificial breeding. But in creatures living in the free state, mutations arise pretty often. There too, then, idiokinetic influences must be effective.

Especially, in this connexion, we must think of the effect of certain radiations. The investigations of physicists have shown that our environment is permeated by numerous radiations of the nature of X-Rays and radium radiations, although of course these are not so concentrated as they are in Röntgen-ray laboratories.

Not infrequently the view is expressed that immoderate quantities of mental work, such as are called for in many occupations, lead to degeneration and to dying-out of the family. There is no adequate evidence of any such causal relationship, for the dying-out of families of mental workers can be mainly accounted for by gonorrhoea, syphilis, and deliberate prevenception; and, furthermore, morbific heredity factors may be originated in these families by the unwholesome influences of urban life. Still, I am far from denying the possibility that excessive mental labour may have a directly injurious effect upon the hereditary equipment. We do not know enough about this matter, and, pending further information, we should do well to take the darker possibilities into account. The comparative frequency of morbific taints, on the one hand in the more exalted nobility, and on the other in the Jews, who have for numberless generations been living almost exclusively in towns, certainly tends to support the view that some of the influences of urban life may have permanently injurious effects upon the hereditary equipment.

In these groups (the high nobility and the Jews), many people have been inclined to point to "inbreeding" as a cause of degeneration. I am sure that the hypothesis is unsound. For practical purposes, there never occurs among human beings an inbreeding so close as that which has been shown in animals and plants to weaken the stock (see above, p. 109). In the few instances among human beings where inbreeding is known to have continued for many generations, as in the Ptolemies and in the ruling house of the Incas of Peru, no injurious consequences have been reported.

Miss King[1] inbred white rats for 25 generations, and could not detect any injurious consequences, though she dealt in all with 25,000 individuals. There was, in these, a rigorous selection at work, the strongest and healthiest animals being chosen to breed from generation after generation; but, nevertheless, the experiment showed clearly that long-continued and very close inbreeding does not suffice by itself to lead to degeneration in rats. W. E. Castle[1] inbred the fruit-fly Drosophila very closely for 59 generations without noting any injurious consequences.

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[1] H. D. King, Studies on Inbreeding, "Journal of Experimental Zoology", 1918–1919: vol. XXVI, nos. 1 and 2; vol. XXVII, no 1; vol. XXIX, no. 1.

Obviously the fact that in cases of inbreeding recessive heredity factors are peculiarly apt to coincide and thus to become manifest, has often led investigators to infer that inbreeding in itself is injurious. But there is no such thing as the "ageing" of a family, and there is no such thing as the "rejuvenation" of a family by the "introduction of new blood". The "oldest" of noble families are, biologically considered, no older than any other families. Where individuals are concerned, we cannot attribute every progressive weakness to the effects of old age, and still less can we do so in the case of a family or of a race, in which there never occurs anything like "ageing" from internal causes. All degeneration ensues from the effect of external causes.

Untenable likewise is the widely prevalent view that undue age or youth of the parent can, per se, lead to the production of low-grade offspring. If it be found that the children of very young parents (those under 17 years of age) are in general low-grade individuals, the presumable explanation is that under extant social conditions persons who reproduce their kind when they are so young are themselves of low grade (feeble-minded girls, and the like). On the other hand, elderly primiparae, whose children (we are told) are often of low grade, are likely themselves to be of low grade—or they would have mated and had children earlier; and elderly fathers (especially men who marry late in life) have much more often had syphilis than have young fathers, and have been much longer exposed to all kinds of idiokinetic noxious influences (alcohol, tobacco, etc.). There is no satisfactory evidence in support of the view that age as such in the parents has an injurious influence upon the offspring.

Similar considerations apply to the alleged "inferiority" of firstborn children. It is certainly conceivable that at the time of the birth of her first child a mother may not be as well fitted for her task as she afterwards becomes; but this can have no effect upon genuinely hereditary characteristics, and the slightly lower weight, on the average, of firstborn children is usually made good ere long. The statistical evidence offered in support of an "inferiority of firstborn children" in respect of mental disorders, short sight, tuberculosis, etc., will not stand criticism. It is only because there are more firstborn children than secondborn, thirdborn, etc., that inferior specimens are somewhat more often firstborn than secondborn, etc. Contributory to the same phenomenon is the circumstance that sickly families on the average produce fewer children than healthy ones.[1] Besides, such troubles as mental disorder, short sight, and tuberculosis, which only develop as life goes on, have often made their appearance in the firstborn when in the younger brothers and sisters the taint has not yet become manifest. When in families containing the same number of children, persons of identical ages have been compared, there have been no signs of any such "inferiority of firstborn children".[2]

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[1] Genetics and Eugenics, Cambridge, Mass., 1922, p. 230.

A favourite theory is that degeneration arises above all from under-nutrition, and could therefore be avoided if people had more to eat and to drink. It is unquestionably conceivable that the lack of certain essential nutrients may injuriously affect the upbuilding of the hereditary equipment. But it is a very different question whether in actual fact under-nutrition plays a part as a cause of hereditary modifications. There are no satisfactory grounds for any such supposition. However much we may deplore the sufferings of the population of Germany from an inadequate supply of nutriment during and after the war, there is happily no reason whatever for the belief that racial degeneration has ensued. In such matters, people devoid of critical acumen are far too ready to be influenced by political, moral, or other emotional considerations. For like reasons, the hereditarily injurious effects of alcohol and syphilis have often been exaggerated, whereas people have for emotional reasons been inclined to deny that X-rays and medicaments can injure the hereditary equipment. There is a widespread inclination to regard degeneration as the outcome of immoral behaviour, and to look upon all things which can be considered morally unexceptionable as harmless in every other respect. Nature, however, knows nothing of our moral valuations, and is concerned with necessity alone. Nebulous Lamarckian theories are apt to prompt the arguments of those who maintain that degeneration may arise from under-nutrition, overwork, etc.—states of exhaustion resulting from such external noxious influences being without warrant assumed to be inheritable.

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[1] A. Ploetz, Neomalthusianismus und Rassenhygiene, "Archiv für Rassenbiologie", vol. X, nos. 1 and 2.

[2] Cf. W. Weinberg, Ueber die Frage der Minderwertigkeit der Erstgeborenen, "Oeffentliche Gesundheitspflege", 1916.

Closely akin to the Lamarckian superstition is the belief in what are called "maternal impressions". If, for instance, a child is born with a hairy birthmark, we shall be told that the cause was that the mother was frightened by a mouse during pregnancy. In like manner, port-wine marks in the child are supposed to have appeared because the mother saw a house on fire—or, lacking a convenient conflagration, because the mother, when pregnant, saw a plate of strawberries. If the child is unusually ugly, the mother, when pregnant, must have seen a monkey. And so on, and so on—the supposedly causal incident being in most cases retrospectively touched up. Malformations in the newborn child are often referred to the sight of a mutilated person by the pregnant mother, or sometimes to a fright experienced by the mother. If a child is inclined to be tearful, this is supposed to be because the mother wept much during pregnancy—whereas the real connexion may well be that both mother and child have inherited a family tendency to be lachrymose.

Since the doctrine of "internal secretions" has become fashionable, we have often been assured that hereditary modifications must have arisen in this way—which is regarded as piquant.[1] The readiness with which laymen are apt to adopt such views uncritically and amateurishly is in inverse proportion to their capacity for forming any clear ideas of the processes in question. The actual connexion between hereditary equipment and the processes of internal secretion is, not that the internal secretions modify the hereditary equipment, but, conversely, that the organs of internal secretion themselves originate as parts of the hereditary equipment. As Morgan puts it, internal secretion is one of the ways in which the hereditary equipment takes effect.[1]

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[1] Cf. A. Elster, Sozialbiologie, Berlin and Leipzig, 1923.

Widely diffused is the superstition that earlier impregnations have a lasting effect, and the imposing word "telegony" has been coined to denote this supposed working. It is said that a pedigree bitch, having once been crossed by a mongrel and having given birth to mongrel offspring, will no longer be able to produce pure-bred offspring when crossed by a dog of her own race. If such an after-effect were really possible, hereditary diseases could of course be diffused in a similar way, and such absurdities are actually declared to have taken place. Trustworthy experiments on animals have shown, however, that "telegony" is a mere superstition, obviously dating from the days when people did not yet know that fertilisation is effected by the entrance of a spermatozoid into an ovum. Before the rise of the modern doctrine of heredity, it might well seem utterly inexplicable how, from the union of two outwardly similar parental animals, offspring of an extremely different aspect might sometimes issue. To-day, however, when we find that, for instance, a wild-coloured doe rabbit, having been covered by a wild-coloured buck rabbit, may give birth to white or to black offspring, we know that a masked heredity factor has come to light. In such cases, however, what we see is not the after-effect of previous fertilisations of this doe rabbit, but the after-effect of alien fertilisation in previous generations. The superstition in question was the main foundation of an antisemitic sensational novel which was circulated by hundreds of thousands shortly after the war. The author's implication is that thanks to such after-effects the German people is being more and more "poisoned" by Jewish blood.[2]

In many cases a change in the hereditary equipment appears to depend upon nothing more than a disturbance of the antagonism of homologous genes. As was explained in the first section of this book, the general rule is that in the formation of the reproductive cells two homologous genes separate. But there are exceptions. Thus C. B. Bridges was able to show[3] that in exceptional instances two homologous chromosomes enter the same reproductive cell, or both may fail to enter such a cell. Apparently, the same thing can happen to parts of the chromosomes. When in the course of the reduction division the chain of chromomeres (see above, p. 74) in two homologous chromosomes does not divide at precisely the same spot, the consequence will be that one chromomere will pass to the "wrong" side. In this way there will be produced reproductive cells devoid of this particular chromomere, and others containing it twice over. Such a lack of a chromomere or a gene will have exactly the same result as the destruction of a gene by chemical or physical influences; that is to say, there will thenceforward be a recessive defect in the hereditary equipment. It is, indeed, far from improbable that the majority of variations by defect are the outcome of such a disturbance of the antagonism of the genes. When the maturation of the reproductive cells takes place under exposure to the influence of idiokinetic factors (for instance, such narcotising substances as alcohol), the straightforward exchange of the chromomeres will, of course, be especially liable to slight disturbances.

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[1] T. H. Morgan, Human Inheritance, "American Naturalist", vol. LVIII, 1924, no. 658.

[2] A. Dinter, Die Sünde wieder das Blut.—In the "Archiv für Rassenhygiene", vol. XIII, 1920, nos. 2 to 4, p. 227, I have pilloried the way in which Dinter unwarrantably invokes the authority of the German Society for Racial Hygiene.

[3] Non-disjunction of the Sex Chromosomes of Drosophila, "Journal of Experimental Zoology", 1915.

Such a disturbance of the antagonism would, it need hardly be said, lead just as often to the doubling as to the absence of a gene; and we may presume that by such a doubling of a gene in a hereditary equipment there may be caused the first appearance of polymeric factors, that is to say of several homogeneous factors, in one hereditary equipment.

The easiest to understand are those hereditary modifications which arise from the simple lack of a gene; and the next most intelligible are those which depend upon an accumulation of like genes. But in addition to these comparatively quantitative changes there must also be strictly qualitative changes as the outcome of the accumulation or modification of molecules within the genes.

Idiokinesis does not result only in the production of new morbific heredity factors; for we must also regard as having been idiokinetically originated all those countless hereditary modifications which have rendered possible the phylogenetic development of a living organism. But this phylogenetic development or racial evolution can only have been effected through the cooperation of a rigorous process of natural selection, leading to the preferential survival of those exceptional mutations which have conferred increased power of adaptation. Under extant conditions, since as far as the human species is concerned no such rigorous selection is going on, idiokinesis is for practical purposes mainly deleterious in its effects. Inasmuch as the loss of heredity factors is much commoner than the origination of new ones, and inasmuch as most dominant mutations are not of a kind to increase the power of adaptation, the net upshot of idiokinesis is a progressive deterioration of the hereditary equipment and an intensifying degeneration. Furthermore there may be a reversed selection, a "dysgenic" selection, leading to an increase in the degeneration, as will be shown in the sequel to the present work. However, let me insist once more that heredity, far from being a doom, is rather to be regarded as a source out of which all life flows.

Whereas the notion of illness or disease applies to a more or less transient restriction of the capacity for adaptation as well as to a permanent restriction of that capacity, the concept of degeneration is limited to hereditary trends. Thus a population may be thoroughly permeated by non-hereditary diseases (such as syphilis)[1] without being for that reason degenerate. When we speak of degeneration we mean the origination and diffusion of morbific heredity factors, so that the descendants lack the hereditary efficiency of the ancestors. Degeneration is the central problem of racial hygiene, and the prevention and the overcoming of degeneration are its main objects.

Since the publication of the first edition of this book, I have been asked how I can account for the fact that hitherto there have not been directly observed any "progressive" hereditary modifications such as would entail an increase in the capacity for adaptation—although such modifications must certainly, though rarely, arise. I account for it in this way. Mutations whose appearance can be directly observed are, for the most part, marked deviations from the previously extant type, and it is self-evident that such great jumps must for the most part be unfavourable to self-preservation. For this reason, racial evolution takes place mainly by small steps, by mutations which do not differ markedly from the pre-existent type. Now, even if we could readily recognise such slight mutations in human beings, we should hardly be in a position to say whether they would really entail an increase in the capacity for adaptation. A decline in the capacity for adaptation can often be recognised easily enough, but not so an increase in that capacity, for in the latter case we could not decide without a knowledge of the significance of the mutations in the most diversified conditions of existence, and such knowledge is practically unattainable. It seems to me that this difficulty of recognising a "progressive" hereditary modification when it does occur explains why no such modifications have hitherto been observed.

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[1] The lay reader must not forget that although syphilis can and does pass by infection from the mother to the embryo, giving rise to *congenital* syphilis, syphilis is not a *hereditary* disease.—TRANSLATORS' NOTE.

It has been very widely contended, to a great extent even by supporters of the theory of natural selection, that selection can have no significance for the origination and for the nature of mutations. I should like to take this opportunity of expressing my dissent. We are agreed that the nature of mutations is very largely determined by the pre-existing constitution of the hereditary equipment. But by selection from among the various biotypes of a population there is produced the soil in which mutations can arise, and inasmuch as their nature is more dependent upon the nature of this soil than upon anything else, the nature of mutations will perforce be determined in some measure by selection.

For a considerable time the opinion prevailed that the hereditary equipment is extremely stable, is modifiable with great difficulty, and that mutations are too rare to have a decisive bearing upon degeneration. I myself have never shared this view, and in a work published in 1912 I declared that mutations are obviously "extremely common", and must therefore play an important part in bringing about degeneration.[1] Recently Baur, likewise, has declared that mutations are "extremely common". He estimates that in the snapdragon (Antirrhinum) at least 10 % of all individuals are carriers of mutations. An estimate made by H. J. Muller and E. Altenburg,[2] two of Morgan's collaborators, is almost identical. These observers found that in the fruit-fly (Drosophila) each gene must, on the average, undergo a mutation every two thousand years. Calculation shows that in that case about 8 % of all individual fruit-flies must be the carriers of mutations. Morgan, himself, has declared that the hereditary equipment is somewhat inconstant. If this be so in such organisms as the snapdragon and the fruit-fly under the natural conditions of existence, although their structure is relatively simple, how much commoner must mutations be in the highly complicated hereditary equipment of human beings, exposed as they are to the most diversified conditions, many of which are extremely unnatural! But hereditary modifications anything like as extensive as this, occurring in the absence of rigorous selection, must necessarily within a very few generations lead to extensive racial degeneration. This conclusion is logically irrefutable.

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[1] Ueber die krankhaften Erbanlagen des Mannes, Jena, 1912.

[2] The Rate of Change of Hereditary Factors in Drosophila, "Proceedings of the Society for Experimental Biology and Medicine", vol. XVII, 1919, p. 10.

SECTION FOUR

METHODOLOGY

by

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CHAPTER ELEVEN

METHODS FOR THE STUDY OF HUMAN HEREDITY

INASMUCH as we are not in a position to study human heredity by experimental breeding, the theory of the subject is, as far as generalities are concerned, based upon analogies drawn from experiments on plants and animals, and, when we come to details, upon statistical methods. Of course the value of reasoning by analogy has its limits, but these limits are not so narrow as is often assumed. Laws found to be equally valid in their application to peas and snapdragons, flies and butterflies, farmyard fowls and canaries, mice and rabbits, are obviously applicable to human beings as well, for man is much nearer, not only to rabbits and fowls, but also to flies and butterflies, than to plants.

Thus the application of the mendelian law to human beings is fully justified by analogy. We are entitled to say that *the human hereditary equipment, likewise, consists of distinct heredity factors or genes which are substantially or materially present in the reproductive cells; that in the course of generations these genes undergo disjunction and become reassociated while retaining their individual peculiarities; and that any two of these genes behave in such a way that during the formation of the reproductive cells they hardly ever enter the same cell but in the great majority of instances enter distinct cells, so that each of them has an equal chance to participate in the making of a particular child.* The numerous facts of direct experience concerning the inheritance of human trends harmonise perfectly with the above generalisation; and, on the other hand, there are no known genealogical or statistical data which conflict with the belief that the mendelian law is applicable without exception to human beings. There is no analogy in the human species to the non-mendelian inheritance supposed to have been proved in plants (transference of chromatophores, etc.).

From experiment and observation on animals and plants we are also entitled to conclude that in man (as in other animals and in plants) there is no such thing as the "inheritance of acquired characters". It would be utterly absurd to suppose that human beings in this respect can differ from other animals.

It is true that a noted clinician has maintained that, whereas in animals the inheritance of acquired characters may occur, the human species is now "stabilised". The occurrence of manifold morbific heredity factors (which have certainly not all existed ever since man became man) suffices, however, to prove that the human species has not been "stabilised"; and, for the rest, the non-hereditability of acquired characters is by no means synonymous with the stabilisation of a species (see above, pp. 465 et seq.). On the other hand, an opposing view is not infrequently voiced, for we are told that, whereas in the case of plants and in that of other animals than man, the inheritance of acquired characters has not been proved to occur, in human beings the inheritance of acquired characters must take place, for otherwise no progressive civilisation would be conceivable. Those who voice such views show a lamentable failure to understand the difference between biological inheritance, on the one hand, and the handing-down of the acquirements of civilisation, on the other. The truth of the matter is that as regards human beings, too, there is no trustworthy evidence in favour of any such inheritance of acquired characters, whilst there is a great abundance of evidence conflicting with the hypothesis.

Reasoning by analogy from experiments made on other animals, in conjunction with observations concerning sex-linked inheritance, has thrown light upon the determination of sex in human beings likewise. This matter has already been discussed. The leading authorities, S. Painter,[1] K. Oguma, and H. Kihara,[2] tell us that the number of the chromosomes in human beings is 48 in diploid cells (fertilised ovum, body cells) and 24 in haploid cells (reproductive cells). Until recently, the numbers were usually given as 24 and 12 respectively. The study of chromosomes in human beings is very difficult, because the conditions are extremely unfavourable for microscopic work and suitable material is hard to obtain. The before-mentioned investigators believe that in the human male, also, they have been able to detect heterozygotism of the sex chromosomes. According to Painter, in men there are

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[1] Studies in Mammalian Spermatogenesis, "Journal of Experimental Zoology", vol. XXXVII, 1923.

[2] Oguma and Kihara, Etude des chromosomes chez l'homme, "Archives de Biologie", 1923.

X and Y chromosomes; but according to Oguma and Kihara, only an X chromosome. Either would conform to theoretical expectations. We may ask ourselves, indeed, whether these theoretical expectations may not have influenced the observers in the interpretation of microscopic appearances that are by no means unambiguous. However this may be, the microscopic study of the human reproductive cells has not told us anything new about human heredity. Cytology (the study of cells), which is of great importance to our general knowledge of heredity, cannot be the main foundation of our study of human heredity.

Passing from the general laws of heredity to consider matters of details, we cannot expect that we shall always find the same close correspondence between man and other animals, and we have therefore been led to make a special study of such matters in human beings. The impetus for the attempt to elucidate the determination of sex in human beings came originally, not from discoveries made in the case of the fruit-fly (in which sex determination is upon the same lines as in human beings), but from researches on butterflies, in which the female sex and not the male is heterozygotic. But the very fact that the conditions in butterflies and in human beings respectively were conversely analogous helped to show the way to the solution of the problem as far as human beings were concerned. In other instances, however, the analogy is much closer. Thus the heredity of complete albinism in human beings is precisely identical with that of the same condition in many other animals, being simply recessive. Since, however, we cannot tell beforehand how close the analogy will be, it is essential in every case to study the available statistical material relating to human beings.

It follows that the main value of reasoning by analogy is that we are thereby enabled to draft plausible hypotheses, and these must be sifted in the light of observations directly made on human beings. In default of the knowledge we have obtained concerning the determination of sex in other animals, the solution of the problem as concerns human beings would have been a much more difficult matter. Still, we cannot go so far as to say that the solution would have been impossible.

For thousands of years, all kinds of hypotheses concerning the determination of sex have been formulated, but one and all of them have been more or less glaringly in conflict with the facts of experience. It remains possible, none the less, that ultimately the right hypothesis might have been hit upon, and this would then have stood the test of confrontation with the facts—especially with the facts relating to sex-linked inheritance.

We see, then, that the progress of science takes place by a selection from among the hypotheses that are apriori possible. These hypotheses are confronted with the results of experience; those which obviously conflict with experience are rejected; and the survivors, the "fittest", are thereby rendered more probable. In strict logic, of course, a hypothesis is not yet "proved" merely because it has not been found to conflict with experience. In the empirical natural sciences, there can be no such thing as absolute proof. "When we are interpreting experience, we can never say with absolute certainty: 'This is so.' In countless instances, however, we can say with complete assurance: 'That is not so'." Such are the words of the Bonn mathematician E. Study.[1] When, after a great deal of experience relating to a particular hypothesis has accumulated, the facts have not been found to conflict with the hypothesis in any way, that "hypothesis" becomes a well-grounded "theory". That is why we speak to-day of the "mendelian theory", of the "theory of sex determination", etc.

I know that there are investigators who despise "theory", and who believe themselves entitled to dismiss as "speculative" even the simplest of conclusions so long as these have not been "proved" experimentally or statistically. I should like to ask such critics whether "pure experience" or "pure induction" can, as a matter of principle, furnish any knowledge at all. Pure speculation can furnish knowledge, as we see from the example of mathematics; but pure experience can never do so. In the domain of the natural sciences, knowledge always results from the cooperation of cautious speculation with cautious induction. Even those who tell us that they despise all speculation, speculate unconsciously in order to win results—if their work yields any results at all.

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[1] Denken und Darstellung, Logik und Werte, etc., Vieweg, Brunswick, 1921.

Physics and chemistry, which are held up before the biologists as the exemplars of exact research, have to-day unmistakably entered upon a speculative phase, although observation and experiment (especially the latter) lend invaluable aid. The science of heredity is entering this phase likewise. It will be enough to remind the reader of Morgan's pioneer researches. "As science advances, theory tends more and more to become the central feature of investigation," writes Study. Though it be true that in the science of human heredity the hardening of hypotheses into theories is the result, not of experimental, but of statistical enquiry, this does not mean (as is so often assumed) that there is a fundamental difference here between the study of man and the study of other animals. In physics and chemistry, no less, precise data are registered as the outcome of numerous measurements, into all of which there has entered an element of probable error. Thus qua knowledge, the value of statistics differs from that of the experimental method only in degree, not in kind. Neither method can furnish "absolute certainty", and it is therefore unjust to demand "absolute certainty" from the science of human heredity. But to admit this does not imply the admission that everything in the domain of that science is uncertain. The certainty of the acquirements of the science of human heredity stands on the same footing as that of the acquirements of human biology in general, and especially on the same footing as that of therapeutics and hygiene in general. The number of hypothetical possibilities is not unlimited. The general science of heredity, being based on experiment, does not leave so vast a number of possibilities open; and in the case of human beings our statistical material will often enable us to make the choice from among these possibilities with a certainty sufficient for practical purposes—with a certainty which is by no means inferior to that which guides us in the ordinary decisions of life.

In the general study of heredity, the experimentalist deliberately makes certain crossings; in the study of human heredity, the investigator seeks for similar crossings. Thus Eugen Fischer has systematically studied the offspring of crossings between Hottentots and individuals belonging to other races (see above, p. 136), and C. B. Davenport has carefully studied the mixed breeds containing Negro blood (see above, p. 146). Speaking generally, we may assume that all the crossings which are of practical interest must have been made somewhere or other among the many hundreds of millions of which the population of the world consists. If we can trace them out they will be just as useful to science as if they had been deliberately effected, and to find them ready-made will save us a great deal of time. The same method is indicated for the study of morbid taints. Vogt, for instance, has made a systematic search for families in which the daughters suffered from red-green blindness, and has thus been enabled to draw the most valuable conclusions (see above, p. 259).

The question has often been mooted whether some particular doctrine forming part of the science of heredity, for instance the doctrine of sex determination, is a "fact" or a "theory". When I am asked this, I am wont to counter by enquiring whether the movement of the earth round the sun is a fact or a theory. To this the usual reply is that the movement of the earth round the sun is a fact and not a theory, for people have long since got used to the idea and have come to accept it as a fact. I need hardly say, however, that here we have to do with nothing more than a theory, and that direct experience, the evidence of our eyes, certainly does not teach it us as a fact. Pure observation cannot provide us with any knowledge at all, and therefore cannot provide us with any knowledge of fact. But when we have grasped the truth of Kant's statement that "whatever is factual is already a theory", we shall avoid the mistake of allowing a hostility to all theory to make us prefer to well-grounded theories long-since refuted prejudices in which we fail to recognise the hypothetical element.

The registration of certain facts in accordance with quantity and number, and their elaboration in order to ascertain general rules or laws, constitute the essence of the statistical method. In the domain of the science of human heredity, we must apply this method in two different ways, one of them being comparatively individual-statistical or genealogical, and the other being comparatively mass-statistical or biometric. The individual-statistical study of heredity takes the form of a search for evidence of particular hereditary trends within a kinship. This method is that of family history or of genealogy, whose main object according to Kekule von Stradonitz[1] is "to provide the materials for the science of heredity, insofar as this science is concerned with human beings". The materials hitherto available in the form of genealogies are for the most part inadequate for the study of human heredity. In its essential nature, genealogy is a historical discipline; it is concerned with individual matters, with individual persons and families and their interconnexions. But the science of heredity, as one of the natural sciences, is primarily concerned with generalities, with establishing general laws—individual persons and families, as such, being matters of little moment to science. The geneticist wants to know, for instance, how schizophrenia is transmitted by inheritance, and is not particularly concerned to know precisely how schizophrenia appeared in the kinships of the Guelphs and the Wittelsbachs. To geneticists, historical information is only a means to promote the general knowledge of their science. Thus regarded, genealogy can render great services by demonstrating family connexions, and also by supplying reports about the qualities of individual persons; but, in general, the study of family history as it must be pursued by the geneticist is of a fundamentally different type, being scientific family history or family anthropology.[2] The geneticist has to obtain the fullest possible relevant knowledge about all the kindred of the patient whose case gave the impetus to his particular research. A graphic representation of the parents, grandparents, great-grandparents, etc., of the individual from whom an investigation starts (such a person is called a *proband*), is known as that person's *ancestral table.* When, on the other hand, we make a graphic representation of all the offspring of a particular person, we speak of a *descendants' table* or of a *family* or *genealogical tree.* Fig. 157 on page 502 shows an ancestral table. Figs. 32 and 40 show descendants' tables. Various systems of numbering are used to denote the individual persons in an ancestral table or in a genealogical tree. Fuller details regarding this matter will be found in Scheidt's book. When we represent in one and the same table both the ancestry and the descendants of a proband, and perhaps in some cases the collateral relatives as well, we speak of a *kinship table*, but this may also be called a family tree in the widest possible sense of the term. For examples, see Figs. 37 and 78. In all such diagrams, males are represented by a circle to which is attached an arrow pointing obliquely upwards and to the right, whilst females are represented by a circle to which is attached a cross pointing directly downwards. If the sex of an individual is unknown, a simple circle is used. Such is the usual diagrammatic method in Germany and in Britain, but there is a slightly different diagrammatic method used in the United States of America. Here a male is represented by a square, a female by a circle, and a person of unknown sex by a triangle. When this method is used, an ancestral table takes the form shown in Fig. 158. The bodily and mental characteristics of all persons in such a kinship, the healthy as well as the diseased, must be ascertained as fully as possible—at least as regards those characters which

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[1] "Deutscher Herold", 1920, no. 1.

[2] W. Scheidt, Einführung in die naturwissenschaftliche Familienkunde, Munich, 1923.

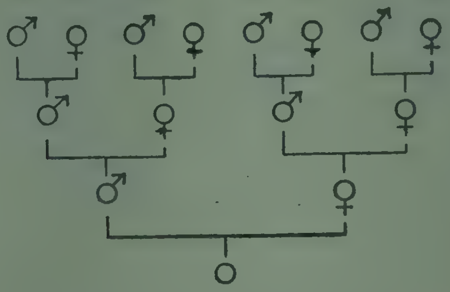

FIG. 157.
Diagram of an ancestral table.

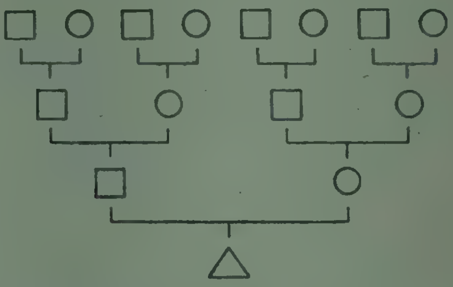

FIG. 158.
The same ancestral table as it would be figured in the United States.

are known to be or may possibly be relevant to the particular research. Kretschmer rightly insists[1] that the study of the inheritance of mental disorders can only be fruitful when all the normal as well as all the psychopathic members of a family are taken into consideration. Whenever we are concerned with morbid symptoms or with other characters which are not already recognisable at birth, but only appear in the subsequent course of life, it is of the utmost importance to record the age of every member of the family at the date of the investigation. Furthermore, for reasons upon which I shall enlarge presently, the investigator should always do his utmost to ascertain whether and in what degree parents are related by blood.

The geneticist must be careful to avoid overestimating the importance of some particular ancestor or ancestors in determining an individual's hereditary constitution. This is a very common error. R. Sommer,[2] for instance, believed himself to have detected a remarkable resemblance between Goethe and a certain Ferdinand Lindheimer, who was related to Goethe through the instrumentality of a great-grandfather's great-grandfather. I cannot consider the circumstance of any notable importance, seeing that this degree of kinship would only make the two men alike in their respective hereditary equipments to the extent of $\frac{1}{1024}$ more than any two members of the general population. On the other hand, those exaggerate who say that we can draw no trustworthy conclusions about a proband's hereditary equipment unless we are informed concerning all his relatives without exception. As regards characters which are mainly determined by only one heredity factor, we can certainly gain much useful information from knowledge concerning a few of the persons who are closely related to the individual under consideration.

The geneticist will not usually find it worth his while to expend much time and trouble upon the search for information about remote ancestors, as is usual in historical genealogy. Knowledge concerning descendants is no less valuable than knowledge concerning ancestors, and information about collateral relatives is equally useful. On the average, an individual has as much of his hereditary equipment in common with that of one of his children as he has with that of one of his parents, and as much in common with a first cousin as in common with a great-grandparent; and, since we can far more readily obtain trustworthy information about the living than about the dead, the study of collaterals is actually more important than the hunting up of information about ancestors. For most purposes it is enough if we try to obtain information regarding a proband's parents and grandparents and regarding all the descendants of the latter. Knowledge concerning cousins may be of the utmost importance, but even more valuable is knowledge concerning brothers and sisters (see below).

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[1] Körperbau und Charakter, p. 94.

[2] Goethe im Lichte der Vererbungslehre, Barth, Leipzig, 1908.

The characters whose heredity has to be traced may be classified as morphological, physiological, and psychological respectively. For the purposes of genetic research, it is of no particular moment whether the psychological characters are looked upon as a subclass of physiological characters in general, or whether they are contrasted with physical characters in general. However this may be, special methods have to be applied to the ascertainment and description of the characters in the three respective groups. I cannot, in the present work, go into the matter at any length, for the methodological differences are not peculiar to genetics, being equally familiar to all students of morphology, physiology, and psychology. Within each department—that of morphology, physiology and psychology—characters are subdivided into healthy and morbid; and for the ascertainment and description of the morbid characters, the investigator must employ the methods of pathology and those of the various clinical specialties. When the assistants to whom the study of family history has been entrusted (in America they are spoken of as "field workers") are not adequately informed regarding the matter of study, the value of the whole investigation is, of course, impaired. A great many illnesses and anomalies can only be rightly appreciated by medical practitioners who have made a special study of them. Consequently, the study of hereditary diseases must mainly be the work of medical specialists. Since these, however, as a rule lack expert knowledge in the domain of genetics and in that of statistical method, the best way is for a geneticist and a medical specialist to collaborate. We must not, however, impose unduly rigorous conditions in such respects. A good many hereditary disorders can be satisfactorily recognised by ordinary general practitioners; and not a few of them even by lay workers, whose talents for the study of heredity may be of a high order. We could never dispense entirely with lay help.

When once the relevant data have been collected (and the mere collection of data is not really part of the science of genetics), the methods whereby they are to be elucidated are identical in all fields. There cannot, for instance, be methods of genetic research peculiar to the psychological field, and so on, although the contrary view has been maintained in a great handbook of biological methodology.

Not infrequently, the data relating to a single kinship suffice to throw light upon the heredity of a disease or of some other character. Especially is this true of those characters which in practice are found to be mainly determined by one particular gene. Although it be true that, strictly speaking, every organ and every character are dependent upon the sum of all the genes, and, conversely, that every gene must exercise some influence upon all the organs and all the characters, we are often able to infer the working of one particular gene from the presence of one particular character. Such characters are spoken of as *monomeric*, in contradistinction to the *polymeric* characters in whose production there has manifestly cooperated a plurality of genes.

As an instance of the way in which data concerning one particular kinship may suffice to elucidate the heredity of a particular character or condition, I may refer the reader to the great genealogical tree drafted by Nettleship to show the heredity of night-blindness (see above, pp. 252 and 253).

Some medical geneticists are so greatly impressed with the importance of the statistical method that they actually regard genealogical research as unscientific, and even think it necessary to apologise if they publish a genealogical tree. This is a most unfortunate attitude, for genealogical research has been and will always remain the foundation of the study of human heredity.

As a rule, indeed, it is by no means an easy matter to ascertain the heredity of a character. If we were at least able to establish definite numerical ratios as between the descendants of persons equipped with particular heredity factors, the mendelian law would naturally enable us to draw valuable conclusions concerning hereditary determination. In the case of human beings, however, the number of children born in any one family is too small for the ascertaining of trustworthy numerical ratios. The genes present in the respective hereditary equipments of the parents combined, in accordance with the mendelian law, quite fortuitously. Inasmuch as for each child of a particular pair of parents the probabilities of any particular combination of trends are equal, it would be possible if we were dealing with a sufficiently large number of children to obtain definite numerical ratios. But when the number of children is small this source of fallacy plays too great a part.[1] In order to reduce the fallacy of inadequate numbers to inconsiderable proportions, we must therefore lump together the data collected from a plurality of families. When we do this, we pass from individual statistics to mass statistics.

Thereby, however, various possibilities of error are introduced. First of all, we must see to it that our data have not been subjected to a one-sided selection, for otherwise they will be of an exceptional and not of an average character. We often find that "interesting" cases of inheritance have been published, cases in which a strikingly large number of persons in one family have been affected by an anomaly, although the anomaly in question may not be caused by heredity at all but may be the outcome of accidental influences. Should we try to deduce numerical ratios from such data as these, they would, of course, be untrustworthy. It follows that families in which there are a few instances only of some particular character are of less value to the geneticist than families in which there are a great many instances. That is a point which must be taken into account during the collection of data, for mistakes made at this time cannot subsequently be compensated.

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[1] It has even been contended that the small number of children in the human family makes the mendelian law inapplicable to human beings; but of course this is absurd. In other animals and in plants the mendelian figures are only the expression of a combination of probabilities—of the probabilities concerning the hereditary constitution of each descendant as determined by the particular constitution of the parents. Now, this applies with equal force to human beings. It is true that the validity of the mendelian law cannot be directly established by the study of data concerning the human species, which can only show that the law has a certain degree of probability. As far as human beings are concerned, the mendelian law can only be shown to be valid deductively, as an inference from the general principle that it applies to all living beings propagating themselves sexually. But this proposition has itself been established by the study of innumerable data relating to the most diversified living creatures.

Above all, we must be careful to avoid jumping to the conclusion that "heredity can have had nothing to do with" the production of a character which, as far as the records show, has appeared once only in a family. A hereditarily determined disease does not, in human beings, necessarily manifest itself in more than one member of a family. If there are not more than from four to six children, we shall generally find, especially in the case of recessive disorders, that only one child suffers.

When we are studying the hereditary determination of a particular disease, we usually begin with all the cases of that disease which have been treated at a particular clinic or by a particular physician, and proceed to enquire whether and to what extent the relatives of the patient have suffered from the same illness or from allied morbid conditions. Of primary importance is the study of the brothers and sisters, the parents, and the children (if any) of the patient. We must remember that, generally speaking, a family is more likely to become the object of study in proportion as the number of the members suffering from illness is larger; for, the more sick persons there are in a family the greater the probability that one or more of them will apply for treatment. Consequently, if we were simply to number off all the sick brothers and sisters and to estimate the ratio of these to the number of the healthy ones, we should obtain an unduly large proportion of sick persons. But this fallacy can be compensated by the methods introduced by W. Weinberg,[1] known as the *brother-and-sister*

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[1] Ueber Methode und Fehlerquellen der Untersuchung auf Mendelsche Zahlen beim Menschen, "Archiv für Rassenbiologie", vol. IX, 1912, Auslesewirkungen bei biologisch-statistischen Problemen, Ibid., vol. X, 1913. Weinberg's presentation of the method is, however, so obscurely worded

method or as the *sib method*,[1] which in its more generalised form is known as the *proband method.* The brother-and-sister method or sib method is based upon the conviction that for the sibs of a proband (the individual from whom an investigation starts) the probabilities as to the combination of parental genes are the same as they are for the proband himself or herself—but that the sibs have not been exposed to a one-sided selection in the way of an accumulation of cases of illness. If, therefore, we draft our ratio from among the sibs excluding the probands, then we shall approximate to the true ratio (the fallacy of small numbers apart). The proband method is wrongly supposed to be difficult to apply. In reality it is based upon the very simple considerations just explained, and its application is easy enough when once the relevant data have been collected. If in any one family there are several probands (if, that is to say, several sick persons in one family present themselves for treatment, so that the sick have not to be discovered by a special enquiry), then each proband must be taken into account also as a sib, and this just as often as there are additional probands besides himself or herself.

It is true that in practice the proband method frequently encounters a great difficulty, whereby its applicability is notably restricted. This difficulty arises out of the circumstance that morbid conditions closely resembling one another clinically and not with certainty distinguishable each from the other may arise from the working of two fundamentally distinct genes. For instance, the group of the hereditary muscular atrophies comprises several biologically distinct conditions (see above, pp. 406 et seq.). It is certain that in addition to dominant taints there are sex-linked recessive taints and simply

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that his book cannot be recommended to the beginner.—An attractive account of it is G. Just's Der Nachweis von Mendelzahlen bei Formen mit niedriger Nachkommenzahl, "Archiv für mikroskopische Anatomie", 1920 (with bibliography of Weinberg's writings); excellent, likewise, is E. Rüdin's Zur Vererbung und Neuentstehung der Dementia Praecox, Springer, Berlin, 1916.

[1] The word "sib" or "sibling" is coming into use in genetics in the English-speaking world, as an equivalent of the convenient German term "Geschwister", and as a general name for all children born of the same parents, that is to say, to denote brothers and sisters without distinction of sex.—TRANSLATORS' NOTE.

recessive taints which can produce the clinical picture of muscular atrophy. Clinically, however, these various forms cannot, as yet, be distinguished with certainty. Consequently, if we were to lump together data concerning muscular atrophy when these data had been derived from the study of various families, we should naturally make seriously false inferences. To use an analogy, we are in many cases of disease not yet able to distinguish the blindworm from the snake. But in the systematic study of the hereditary diseases, just as in the systematic study of biology, we must be guided, not by external, not by phenotypical similarities, but by an inward kinship.

That is why conclusions drawn from the summation of data obtained by the study of different families are so often fallacious. Just as we have to be extremely cautious in reasoning by analogy from animals to man, so likewise must we be very sure of our ground in the study of human heredity before we argue from one family to another. That is why the study of particular family trees will always retain its place in genetics side by side with the study of mass statistics. One trustworthy family tree will sometimes throw more light upon the heredity of a morbid condition than a vast accumulation of statistics. No doubt genealogical study really falls within the domain of statistics, for it likewise consists in the establishment of numerical and metrical ratios, and it, like statistics in general, is concerned with the discovery of general laws concerning numbers and measurements. Even though a genealogical tree should not have been drafted with the special aim of showing an exceptional accumulation of instances, there can be no objection to the deducing of ratios from a large and thoroughly elaborated genealogical tree. But we must, of course, be careful in every case to calculate the average fallacy of small numbers, as is essential in all percentages which are to serve the purposes of comparison.

The usual method is to calculate the mean quadratic error, to expound the theory of which would take me too far afield.[1] I may, however, give a brief account of the formula and its applications. If among n individuals p % exhibit a certain character and the remainder $(100 - p)$ % do not exhibit it, then the mean quadratic error is $\sqrt{\frac{p(100 - p)}{n}}$ %. To give an example: if among a great number of individuals 50 % have a character and the remaining 50 % not, and we select 100 individuals at random, then we may expect to find that of these 100 individuals a fixed percentage with a mean quadratic error of $\sqrt{\frac{50 \times 50}{100}} = 5$ % will exhibit the character. We usually take into account the possibility that the actual error may be thrice the calculated mean quadratic error, and we shall therefore in the instance under consideration reckon with the likelihood that the deviation from the theoretical ratio may amount to 15 %. The possibility that among 100 individuals the number actually found to exhibit the character will not lie somewhere between 35 and 65 is only 1 : 370. If we suppose the ratio to be established from among 40,000 cases, the quadratic error would be only $\sqrt{\frac{50 \times 50}{40{,}000}} = 0{\cdot}25$ %, and the probability would be approximately $\frac{369}{370}$ that the ratio established from among 40,000 cases would lie between the limits $50 \pm 0{\cdot}75$ %.

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[1] As an introduction to the study of statistical fallacies I may recommend G. Pólya, Wahrscheinlichkeitsrechnung, Fehlerausgleichung, Statistik, a contribution to Abderholden's Handbuch der biologischen Arbeits-

Wherever practicable it will be better that the mean error shall be calculated, not from observed numerical ratios (which are always subject to error), but from the hypothetical possibilities, among which a selection must be made under the guidance of experience. Let me illustrate this by a rough example. If the general frequency of a character be 10 %, we shall among 10 sibs most often find that one will exhibit it. Not much less often, however, shall we find among 10 sibs that not one exhibits it. But if from the empirical ratio 0 : 10 we were to calculate the mean error, we should find it to be 0. Setting out, on the other hand, from the hypothetical ratio 10 : 100, we should find for 10 cases a mean error of nearly 10 %, from which we should see without going any farther that the ratio 0 : 10 is very well compatible with the expectation 1 : 10. For the rest, this consideration does not apply only to heredity statistics, but also wherever we are concerned to make a choice between possible hypotheses in the light of statistical material —and guidance in such choices is the main object of statistical inquiries.

The question often arises whether the difference between two

methoden, section 5, part 2, no. 7, Berlin and Vienna, 1925.—Another useful work is G. Just, Praktische Uebungen zur Vererbungslehre, Freiburg-im-Breisgau, 1923.

percentages which have been established by the observation of two distinct series indicates a biological distinction between the material of the respective series, or whether it may simply be the outcome of the fallacy of small numbers. In such cases we calculate the mean quadratic error of the difference between the percentages. This is equal to the square root of the sum of the squares of the errors of the two percentages, that is to say $f_d = \sqrt{f_1^2 + f_2^2}$.

A substantial foundation for the difference may with great probability be assumed to exist when the difference between the observed percentages exceeds thrice its mean error.

We have now to consider how, in actual practice, a decision is made as between the various possibilities of the hereditary determination of a character. First of all we have to ask whether, in the case of this particular character, hereditary determinants are of any importance at all. When we have answered this question in the affirmative, we must proceed to enquire whether the character in question tends to run in families.

Here the proband method must be applied. If a character is, generally speaking, common in a population, its frequent appearance in the members of any one family is far more likely to be due to chance, than if, generally speaking, it is a rare character. For instance, H. W. Siemens[1] has shown that the numerous family trees relating to left-handedness which have been drafted by Jordan and others can by no means serve to prove the hereditary nature of left-handedness, seeing that this anomaly, being extremely frequent, must tend in many instances to run in families. If the general frequency of a character is, for example, 1 : 1000, then a frequency of 1 : 10 among brothers and sisters will point to the cooperation of hereditary trends, provided we are not concerned with a character which may run in families because of joint exposure to the exciting causes of disease or other external influences capable of producing the character in question. When joint exposure to external influences may account for the phenomena, it becomes a very difficult matter to ascertain the significance of hereditary predisposition. That is why there is still so much dispute to-day as to whether heredity does or does not play an important part in the causation of tuberculosis. If only a few members of a family suffer from a disease, and this to a marked extent, while other members of the family are free from it, then (despite the still prevalent opinion to the contrary) we may infer with a high degree of probability that the illness owes its causation in great part at least to heredity. When, notwithstanding the most careful enquiry, we can find no evidence of the operation of external causes, and when determination by these causes even seems improbable, we must always think of hereditary determination. What is not due to environmental influences must be due to the hereditary equipment, seeing there is no third alternative. In many instances we can infer the working of heredity from the nature of a character; as, for instance, in the case of malformations affecting both sides of the body in like manner, or exhibiting a serial homology (affecting hands and feet, etc., alike). If in a child we find that both hands have the same two fingers grown together, and that one of the parents exhibits the same malformation, no one will doubt the soundness of the inference that the anomaly is hereditary. In other instances, conversely, the establishment of the mere fact of hereditary determination may be so difficult that such determination will remain open to question until a particular hereditary course can be shown to be probable. On the other hand, we must not be in a hurry to conclude that heredity has not been at work because in a family or in several families only one instance of a character turns up. This is a point on which I have had to insist more than once.

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[1] Ueber Linkshändigkeit, "Virchows Archiv", vol. CCLII, 1924, no. 1.

When a character is determined by only one gene, we may expect, as a rule, to find the character also in one of the parents; for the gene must be derived from the hereditary equipment of one or other parent, and if the gene acting alone is competent to produce the character in question we must expect to find that character in one of the parents. Such a character, whose origination is due to the action of a single gene and which makes its appearance in every individual whose hereditary equipment contains that gene, is in the science of genetics said to be *dominant*. If the dominant heredity factor be denoted by A then the carrier of a character will usually have the formula *Aa*. Strictly speaking, the idea of dominance implies the assumption that a dominant gene manifests itself in the heterozygotic state as well in the homozygotic. In actual fact, however, with very few exceptions, the morbid heredity factors described as dominant in human beings are known only in the heterozygotic state, and it is not improbable that in the homozygotic state many of them would manifest themselves in a very different way—and in one far more gravely morbid. In such instances, then, we should have to do, not with genuine dominance, but with intermediate or perhaps with interferent behaviour. By *intermediate* behaviour we mean that in the heterozygotic state a gene manifests itself less vigorously than in the homozygotic state, but manifests itself in the same direction in both; whereas by *interferent* behaviour we mean that the direction as well as the intensity of manifestation are different in the heterozygotic and in the homozygotic states respectively. Since, however, in practice such heredity factors in human beings seldom or never manifest themselves homozygotically (because an individual suffering from a hereditary illness or anomaly seldom mates with a person suffering from the same illness or anomaly), the distinction between genuinely dominant, intermediate, and interferent heredity factors is not of much practical importance in the science of human heredity. All three varieties of heredity, which are usually indistinguishable in practice. may be classed together as *covering* heredity factors, or as factors that are "dominant in the more extended sense of the term".

As a rule there is no difficulty in recognising such covering heredity factors, which can often be detected during the study of one single genealogical tree. Of the parents of the carrier of the character, one usually exhibits the same character; and of the children of such parents, half on the average are found to exhibit it.

Not infrequently, however, family trees are published in which a character, although its general behaviour is that of a covering character, nevertheless skips one or more generations. Sometimes this skipping is apparent only, and faulty observation has been at work. If, however, skipping really occurs, we can explain it in the following way. Many morbific heredity factors do not begin to manifest themselves until middle life or old age is reached, and if an individual harbourer of the taint dies before the outbreak of the malady, that individual appears as normal in the genealogical tree (see above, under cataract, p. 243). Secondly there are many factors which do not manifest themselves until certain external influences come into operation. If, for one reason or another, these influences are in abeyance, it naturally seems as if one or more generations had been skipped. In the third place, the manifestations of certain morbific heredity factors may be inhibited by certain other heredity factors. In such cases the appearance of the trouble really depends, not upon one but upon several heredity factors, so that we have here to do with polymerism, which will be further considered below (see pp. 527 et seq., and p. 534).

Among the dominant heredity factors, a special place is occupied by the *sex-linked dominants.* These are heredity factors which are not present in one of the ordinary chromosomes but are localised in one of the sex chromosomes. Rare factors of this kind must exist about twice as often in females as in males, seeing that a woman has two sex chromosomes, a man only one. In some genealogical trees it is not easy to distinguish with certainty between simple dominant heredity and sex-linked dominant heredity. When there is a marked predominance of females among the patients this favours the notion that a dominant factor is sex-linked. Transmission from fathers to sons excludes the notion of a sex-linked dominance, seeing that a man derives his sex chromosome exclusively from his mother. In actual practice, dominant sex-linked heredity plays a subordinate part as contrasted with recessive sex-linked heredity. Only in a few instances, hitherto, has dominant sex-linked heredity been proved or probable (see pp. 277 et seq.), and even then the dominance has been incomplete. If the female carriers of a sex-linked heredity factor exhibit the character in a much weakened form, we speak of *intermediate sex-linked heredity.*

If, in view of the consideration discussed in previous sections of this work, we can exclude the hypothesis that a character has been determined by a single dominant factor (and this is, as a rule, easy enough), the next hypothesis to consider, the simplest and most obvious one, is that of recessive heredity. Easiest to recognise is *recessive sex-linked heredity*, such, for instance, as is known to account for red-green blindness (see above, p. 257). Recessive sex-linked heredity factors can manifest themselves in males, since male human beings have not a second sex chromosome by which the heredity factor in the tainted chromosome can be masked. In females, on the other hand, the factor is usually masked by the corresponding normal (allelomorphic) factor in the other sex chromosome. In females, the morbific factor can only manifest itself when present homozygotically, that is to say in both sex chromosomes.

Taints which are recessively sex-linked are often confounded with those taints which, from their very nature, can only manifest themselves in males (hypospadias, for instance—see above, p. 297); and also with taints which are subject to a change of dominance according to sex. Until recently, all three of these possibilities were classed under "sex-limited heredity". Following Morgan, however, heredity factors localised in the sex chromosomes are no longer spoken of as "sex-limited", but always as "sex-linked". Furthermore, when we speak of a change of dominance in accordance with sex, we do not mean the same thing as when we say that certain heredity factors can only manifest themselves in the members of one sex. In sheep, we are acquainted with certain heredity factors which, whether they are present heterozygotically or homozygotically, can produce horns in males, but in females can produce horns only when present homozygotically. A heredity factor of this kind is dominant in the male but recessive in the female. On the other hand, a dominant factor leading in the male to hypospadias could not, of course, manifest itself in the female even though present homozygotically. Sex-linked heredity factors can never be transmitted from fathers to sons, and such factors can therefore be distinguished from these others, should any doubt arise. The sex-limitation of a character is often no more than incomplete, this meaning that the character in question does not appear exclusively in one sex, though it appears more frequently in one sex than in the other. In this case, the manifestation of the particular heredity factor is merely favoured by sex. The sex-limitation of a character has really nothing to do with its heredity, for non-hereditary characters can also be sex-limited. Thus the "sex-limited heredity" of which Siemens speaks does not really exist.

For the sake of completeness I may refer to the theoretical possibility that hereditary transmission may run its course exclusively in the male line or exclusively in the female line. It has often been believed that certain traits tend predominantly to pass down in the male line; but though this is theoretically conceivable, sufficient evidence of its actual occurrence is still lacking (see above, p. 289). To account for it we should merely have to assume the working of heredity factors localised in the Y-chromosome. Hereditary transmission exclusively in the female line has been observed in certain morbid conditions of plants, and something of the same kind has been seen in one species of butterflies. Should transmission exclusively in the female line ever be observed in human beings, we should have to think of transmission by the plasma (not by the nucleus) of the ovum. It is, however, improbable that hereditary transmission of this type really occurs in human beings.

Simple recessive heredity is far more difficult to recognise than sex-linked recessive heredity. If the recessive heredity factor be called *a*, then all the carriers of the character have the formula *aa*. Much the commonest of this instance is when both parents of a patient are healthy, the factor existing in both in a masked or covered form. Since it is to be expected that among the children of such parents (*Aa*) only one fourth will be diseased, thanks to the working of the law of probability there will be no manifestation of the disease among the offspring of many parents both of whom are harbourers of the taint; and inasmuch as we cannot ascertain the existence of the taint, in such cases, either in the parents or in the offspring, these families will escape our statistical net. If, on the other hand, we include within the scope of our enquiry all those families in which at least one child becomes affected, we naturally get too high a percentage of patients. It is, however, possible to discover the correct ratio by using the brother-and-sister method. When we are dealing with data derived from one clinic or from the practice of one doctor, or with data collected in some similar fashion, the use of the above-described proband method is indicated. If, on the other hand, there come within the scope of our enquiry all the carriers of a character in a particular population, and in general when we are dealing with homogeneous data, we must proceed as if all the diseased individuals were probands.

It seems desirable to illustrate this method by an analytical example. If when the parents have a particular hereditary constitution the probability that a particular child will be diseased is $\frac{1}{4}$, then, if the average number of offspring be two children, we shall find that on the average only in 1 out of every 16 families will both children be affected. Thus the distribution of healthy and diseased children, respectively, in two-children marriages will be shown by the binomial formula $(1k + 3g)^2 = 1kk + 3kg + 3gk + 9gg$. Consequently, the distribution will be that shown in the following diagram

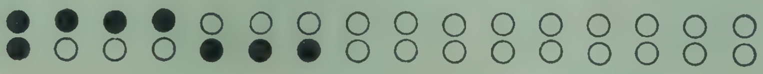

Fig. 159.

Probable distribution of healthy and diseased children in two-children marriages when both parents harbour a masked recessive morbific heredity factor.

(Fig. 159), wherein each pair of circles standing one above the other represents the two children of one marriage.

The ratio between diseased and healthy sibs is 1 : 3, this being in conformity with the recessive heredity. But if those who are collecting data include only the families in which there is at least one diseased child, the last nine families in the table will be excluded from consideration, and we get a ratio of diseased to healthy of 8 : 6, which seems quite out of keeping with the notion of recessive heredity. But the correct ratio can be obtained when the number of the diseased sibs of diseased children is brought into relation with the number of the healthy sibs of diseased children. In the first family (reading the diagram from left to right), each of the two children has a diseased sib (the second is a diseased sib of the first, and the first is a diseased sib of the second). In the remaining six families in each of which there is a diseased child, each of these diseased children has only one healthy sib, this making up six healthy children in all. If we bring into relation with this number, the number of the diseased sibs of diseased children, namely 2, then we get the correct ratio 2 : 6 = 1 : 3, which is in conformity with a recessive heredity involving the probability that on the average one in every four children will be diseased. Corresponding ratios can be worked out for families where the number of children is larger.

But an indispensable prerequisite to the applicability of this brother-and-sister method is, either that in the population subjected to investigation all the families containing carriers of the character under consideration shall actually be included, or else that the data actually subjected to examination shall have a conformable composition. The data must comprise representative families taken at random, not families selected on account of individual peculiarities. The brother-and-sister method in the above-described form is a special variety of the proband method. We can easily see that if it were applied to a collection of data consisting of carriers of the character (probands), it would likewise give us unduly high ratios.

In the foregoing diagram, the first family is twice as likely to be included in an individual choice as any one of the six following families. In an individual choice out of a conformably composed population, therefore, to each family with 2 diseased children there would come only 3 families with 1 diseased child (whereas if a family choice were being made, the ratio would be 1 : 6), and consequently, when the method of individual choice is followed, the above-described brother-and-sister method stil lgives unduly high ratios—in the instance we are now considering 2 : 3, instead of 1 : 3.

The more generalised proband method is therefore preferable in this case. Among the sibs of the proband, in the families 1 to 4, which correspond to an "individual choice", the ratio of diseased to healthy is 1 : 3, this corresponding to the normal probability for recessive taints. Thus on the average one in every four children will be diseased. The term "proband" is drawn from genealogy, having been originally used to denote the individual whose genealogy was to be traced—the individual who formed the starting-point of an enquiry. On the other hand, the carriers of characters, those who only came into consideration because of their kinship with the proband, are termed "secondary cases". Of course the sibs of probands can themselves be also probands, becoming this when they are themselves considered primarily and not secondarily, as when, for instance, they themselves apply to the clinic or to the doctor as patients. In proportion to the completeness with which the carriers of a character in a population come to be treated as probands, does the system of individual choice pass into the system of the complete choice of all families, and the proband method pass into the more specialised brother-and-sister method. Thus the proband method is a generalisation of the brother-and-sister method.

It is often mistakenly supposed that the brother-and-sister method is peculiarly related to the study of recessive heredity. This notion is erroneous. The brother-and-sister method is equally applicable to the study of dominant heredity, being indeed applicable to the study of all those problems of heredity in which we are concerned with establishing a numerical ratio among sibs—for instance, when we have to decide the question whether in many families boys and in many other families girls are born more frequently than corresponds to the general law of probability.

The following calculation made by W. Weinberg[1] shows how great the error can be when the attempt is made to obtain a numerical ratio by the simple counting of the sibs (that is to say, including the probands). Let us assume the true numerical ratio to be $\frac{1}{16} = 6{\cdot}25\,\%$, which would correspond to a dimeric-recessive heredity. Instead of this value we obtain by simple enumeration: if the average number of children in a family be two, 51·6 %; if the average number of children be three, 35·5 %; and if the average number of children be four, 27·5 %. The last figure might readily lead to the erroneous belief that the heredity was simply recessive (when the theoretically calculated percentage would be 25).

Over-enthusiastic advocates of the proband method often assume the so-called "secondary probands". When in the families subjected to investigation there are to be found other series of sibs carrying the character in question, in addition to the series of sibs to which the proband belongs, one member of each of these other series is omitted from the count as a "secondary proband", and only the other sibs are counted. There is, however, no logical justification for such a practice, for these secondary series of sibs have not been subjected to that one-sided selection of starting-point cases for the counteracting of which the proband method has been designed. To the secondary series of sibs, therefore, we must apply the more specialised brother-and-sister method; or, which comes to the same thing, we must regard as probands all the carriers of the character in these sib series. The specialised brother-and-sister method can be unhesitatingly applied to a great kinship whose families have become known through a systematic family research.

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[1] Methoden und Technik der Statistik, a contribution to Gottstein's Handbuch der sozialen Hygiene, Springer, Berlin, 1925, vol. I, p. 132.

Unfortunately, Weinberg's brother-and-sister method is far from providing an ideal solution of the problem we have to solve. All the carriers of a character in sib series containing only one carrier are thereby excluded from consideration; and since the application of the brother-and-sister method comes into account primarily for the study of recessive heredity, or for the study of the cooperation of recessive heredity factors, the majority of the carriers of a character are actually excluded from consideration. Thus the ascertaining of the percentage of the carriers of the character is based upon those carriers who are usually in a minority, but are heaped up in the sib series, so that the error of small numbers becomes comparatively large. It is unquestionably a blemish that the brother-and-sister method, which was designed to obviate the fallacy arising from an aggregation of carriers of a character, should primarily base itself upon sib series in which there is such an accumulation of carriers of a character. It would be well, therefore, to look for another solution of the problem.

The accumulation of carriers of a character arising because sib series containing several carriers of a character are more likely to be included in the material, can be corrected by attaching to the sib series an importance which is inversely proportional to the number of carriers they contain. Of course, each sib series with this reduced importance must be taken into account as many times as the number of probands it contains. We are only concerned to allow due weight for the fact that the "empty" sib series, those which do not contain any carriers of the character, are not represented in the material. We cannot, indeed, determine from our data how many of these "empty" series there are; but we can calculate how many empty sib series may be expected in a particular heritage. Seeing that, in accordance with our general methodological considerations, we are only concerned to ascertain whether the data of experience are or are not conformable to a particular hypothesis, this possibility is sufficient to enable us to form a judgment.

F. R. Berwald[1] was the first to draw attention to the before-mentioned defects in the Weinberg brother-and-sister method. But even though, henceforward, less use is likely to be made of Weinberg's method, geneticists will remain greatly indebted to this authority for having been the first to recognize clearly how it happens that the carriers of a character become aggregated in our material as they do.

If among the sibs of the carriers of a character whose parents do not exhibit the character we find 25 % of carriers of the character, this speaks in favour of a recessive heredity. Among the sibs of the carriers of a character, one of whose parents exhibits the same character, we may expect, if the heredity be recessive, to find 50 % of carriers of the character. But a recessive heredity is not infallibly proved to exist even though both these conditions are fulfilled.

From simply recessive characters (*aa*) which depend upon the coincidence of two homogeneous and allelomorphic heredity factors, we distinguish as *dimeric-dominant* characters those which depend upon the coincidence of two different and non-allelomorphic heredity factors (*B* and *C*). Here carriers of the character both of whose parents are free from the character, usually have the formula *BbCc*; and we may expect to find that 25 % of the sibs will exhibit the character. But in contradistinction to what happens in the case of recessive characters, we may also expect here to find that 25 % of the children of carriers of the character, or a little more than 25 %, will themselves exhibit the character; and that in about half of these sibs, too, one of the parents of the carriers of the character will be a carrier—at least in the case of those characters which do not interfere with reproductive capacity. It is true that in the case of recessive characters the frequency of the simultaneous appearance of the character in parents and children increases with increasing prevalence of the factor in the population at large; but even if the frequency of a recessive character be 1 : 100 (a frequency seldom reached in the case of anomalies), we should only expect to find that the anomaly would be present in about 10 % of instances in one parent of an affected child, and only in about 10 % of the children of an affected parent. In general, the determination of a morbid condition probably results much less often from the coincidence of two different heredity factors than from the coincidence of two like heredity factors. Consequently, most of the anomalies which are found in 25 % of sibs will be simply recessive; but we must always take into account the possibility of dimeric determination.

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[1] Ueber die Weinbergsche Geschwistermethode, "Skandinavisk Aktuarietidskrift", 1924, pp. 75–106.

In the case of rare recessive characters, the corresponding genes will, of course, generally be masked, so that their existence will seldom become manifest. But the commoner a recessive heredity factor is, the more frequently will progenitors who both harbour it come together and thus make it manifest in their offspring. If the frequency of a recessive character be $\frac{1}{n}$, then the frequency of the corresponding heredity factor will be $\frac{1}{\sqrt{n}}$ In this case among the parents and the children of the carriers of the character we must expect to find the character with a frequency of about $\frac{1}{\sqrt{n}}$ (as contrasted with 25 % among the sibs); for all the parents and all the children of a carrier of the character have received the appropriate heredity factor at least once, and another heredity factor of the same kind will tend to coincide in accordance with the general frequency $\left(\frac{1}{\sqrt{n}}\right)$ of the character.[1]

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[1] Since this sentence has been misunderstood (as, for instance, by Dahlberg), who criticises it in his work on twins, let me say in plain terms here, that when I speak of the frequency of a heredity factor it is with reference to the total number of the appropriate allelomorphic heredity factors, and not with reference to the total number of the individuals concerned.

For a knowledge of recessive heredity, the numerical ratio between "sporadic" and "family" cases may also be of importance. In monomeric recessive heredity, a child of heterozygotic parents has one in four chances of being diseased and three in four chances of being healthy. In a sib series of n heads these probabilities therefore combine according to the summands furnished by the binomial $(\frac{1}{4}k + \frac{3}{4}g)^n$ [k represents a diseased child, g a healthy child]. Thus the sum of the probabilities is equal to 1. If, therefore, we wish to reckon the smallest summand as equal to 1—if, that is to say, we wish to ascertain what will be the average number of sib series in which there will be one series containing only diseased sibs—we must develop the binomial $(1\,k + 3\,g)^n$. For instance, in the case of families with four children each, the formula will run as follows:

$$1 \times 4k + 12\,(3k + 1\,g) + 54\,(2k + 2g) + 108\,(1\,k + 3g) + 81 + 4g.$$

Consequently, in every 256 families wherein both parents are heterozygotic, we must on the average expect to find 1 in which all 4 children are diseased; 12 with 3 patients; 54 with 2 patients; 108 with 1 patient; and 81 with 0 patients. We shall thus have to expect 108 "sporadic" cases as against 67 "family" cases. When we are dealing with sibs running more than 4 to a family, the number of sporadic cases will be relatively smaller, whereas when the sibs run less than 4 to a family the number of sporadic cases will be relatively larger (see above, pp. 407–8).

When both parents harbour the same recessive factor, we must expect all the children to exhibit the character. When experience ostensibly fails to justify this expectation we must not jump to the conclusion that the factor in question is not recessive, for it remains possible that the characters in the two parents are not in reality biologically identical. For instance, when we cross two stocks of white fowls in which the whiteness is due to different recessive factors, we may get coloured offspring (see above, pp. 55 et seq.).

When we are trying to ascertain whether a malady is or is not caused by a recessive heredity factor, it may help us a good deal to note the frequency of consanguineous unions among the parents of affected persons. For instance, Hammerschlag found that among 107 children born deaf, 42 (that is to say about 40 %) were the offspring of the marriages of near kin, whereas marriages of near kin form only a small percentage of all marriages, marriages between first cousins being less than 1 % of all marriages. The rarer the character we are studying is in the general population, the more often may we expect to find that persons who exhibit the character are the offspring of the marriages of near kin.[1] If a particular recessive morbific heredity factor existed in a masked form in one family only, the mating of two persons both harbouring the appropriate heredity factor could only occur through the marriage of near kin, so that exclusively in this way could the character become manifest; thus in this extreme case the carriers of the character would all (100 %) be the offspring of consanguineous marriages. If, on the other hand, a recessive heredity factor is widely diffused throughout a population, two such heredity factors will of course often coincide without consanguineous marriage, and the frequency of consanguineous marriages among the parents of persons suffering from the disease will be proportionally smaller. It follows that in the case of very common recessive heredity factors, such as the one which produces blue eyes, we can hardly expect to find that the frequency of consanguineous marriages among the parents of persons exhibiting this character has been greater than the average frequency of consanguineous marriages in the general population. Morbific heredity factors whose heredity is recessive will never, of course, be anything like so generally diffused as this. If, therefore, when we are dealing with extensive material, we cannot find among the parents of persons suffering from the malady we are studying an above-average frequency of consanguineous marriages, we have good ground for inferring that this malady is not caused by recessive heredity factors.

In rural Catholic communes in Upper Bavaria G. Wulz[2] found among 16,000 marriages 0·6 % of marriages of very near kin (ranging up to marriages of first cousins); and in two small country towns among 5000 marriages 0·2 %. During the last few decades, and especially during recent years, there has been a notable increase in the frequency of consanguineous marriages, so that to-day in the rural communes about 0·7 % of marriages are consanguineous. Marriages between relatives whose kinship was not very close were not taken into the reckoning. Studying 453 marriages in three Wurtemberg villages, Spindler[1] found 9 marriages between first cousins (= 1·8 ± 0·7 %), but Reutlinger,[2] studying 117 marriages among Jews in two country towns of Hohenzollern, found no less than 19 marriages of first cousins (= 16·2 ± 3·4 %), and this great frequency in the marriages of very near kin may well explain the comparative commonness of recessive anomalies in the Jewish population. Speaking generally, consanguineous marriages are rarer in Catholic regions than in Protestant, for among Catholics a dispensation has to be obtained before such marriages can be solemnised. Consanguineous marriages are especially common in small communities of those professing some particular faith surrounded by a population of persons professing some other faith—religious "enclaves", as it were. In Prussia, according to official records, marriages of near kin as a percentage of marriages in general were 0·45 in 1907, 0·48 in 1908, 0·44 in 1909, 0·42 in 1910, 0·42 in 1911, 0·40 in 1912. Marriages between second cousins and between relatives less closely connected than this have obviously been excluded from the reckoning. We may doubt, moreover, whether the registrars always inquire as to kinship among those whose marriages are registered. Czellitzer, studying the official records in Berlin during the years 1896 to 1913, found a frequency of marriages of near kin (up to and including the marriages of first cousins) amounting to 0·62 %, the marriages of first cousins alone amounting to 0·57 %. To render exact comparisons possible, it is essential to define up to what degree of blood relationship we go when we speak of consanguineous marriage or the marriage of near kin, but unfortunately such precision has usually been lacking. The consanguineous marriages of grandparents has no bearing on our problem, although this fact, too, has often been overlooked.

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[1] F. Lenz, Die Bedeutung der statistisch ermittelten Belastung mit Blutverwandtschaft der Eltern, "Münchener Medizinische Wochenschrift", 1919, no. 47.

[2] Ein Beitrag zur Statistik der Verwandtenehen, "Archiv für Rassenbiologie", vol. XVII, 1925,, no. 1.

The *systematic study of areas where inbreeding prevails* is of great importance in order to throw light on the heredity of recessive maladies. As E. Hanhart[3] has insisted, such a study should be undertaken everywhere as soon as possible. There are two strong reasons for urgency. First of all, owing to increasing facilities for communication, communities isolated enough for inbreeding to prevail in them are rapidly becoming rare; and, in the second place, owing to the progressive decline in the birthrate, the great kinships requisite for the establishment of trustworthy numerical ratios are becoming more and more infrequent.

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[1] Ueber die Häufigkeit von Verwandtenehen in drei württembergischen Dörfern, "Archiv für Rassen- und Gesellschaftsbiologie", vol. XIV, 1922, no. 1.

[2] Ueber die Häufigkeit der Verwandtenehen bei den Juden in Hohenzollern, "Archiv für Rassenbiologie", vol. XIV, 1923, no. 3.

[3] Ueber die Bedeutung der Erforschung von Inzuchtsgebieten, etc., "Schweizerische Medizinische Wochenschrift," 1924, no. 50.

In studying the heredity of anomalies, we fairly often come across instances in which there are many reasons to believe that the heredity of such an anomaly is recessive, but that among the sibs less than 25 % are affected with it. This failure to reach the expected average may be due to various causes. The first thing we have to ask ourselves is whether the morbific heredity factors, although present homozygotically, may not need to be reinforced by additional influences, operating from without, before they can make themselves manifest. In the second place, it is possible that the cooperation of supplementary heredity factors is essential to the manifestation of the character, this being polymeric. In the third place, the numerical ratio may be modified by the selective dying-out of affected sibs. In the fourth place, the spermatozoids tainted with a particular heredity factor may be less successful in effecting fertilisation than other spermatozoids not so tainted. Heribert-Nilsson uses the term "certation" (competition) to denote such a selective process occurring during the act of fertilisation. (See above, p. 253.) On the other hand, the fertilising capacity of certain spermatozoids might conceivably be intensified by particular morbific heredity factors.[1] It is easy enough to demonstrate selective fertilisation in breeding experiments on animals, but in human beings such selective fertilisation is very hard to prove, and in the case of recessive anomalies the proof is practically impossible.[2] Furthermore since the other possible causes of a disturbance of the numerical ratio are for the most part difficult either to grasp with precision or to exclude with certainty, we must be careful not to attach decisive importance to the demonstration of an exact numerical ratio as proof of the nature of a particular manifestation of heredity. Theoretically, too, we have also to take into account the possibility that in isolated instances the anomaly whose heredity is being investigated may have been the outcome of a recent mutation; and in such instances we naturally should not expect to find an identical anomaly among the sibs.

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[1] G. Hiort, Die ungleiche Bewegungsgeschwindigkeit der Spermatozoen als Ursache der Verbreitung krankhafter Anlagen, "Archiv für Rassenbiologie", vol. XV, 1923, no. 3.

[2] F. Lenz, Ueber den Nachweis selektiver Befruchtung beim Menschen, "Archiv für Rassenbiologie", vol. XVI, 1925, no. 4.

Now let us proceed to consider in somewhat fuller detail how a ratio of less than 25 % might be produced by the cooperation of several heredity factors. A condition may be due to the homozygotic presence, not of one pair of these, but of two pairs. We then speak of *doubly recessive* or *dimeric-recessive* factors. From experiments on animals and plants we have become acquainted with a number of such dimeric-recessive factors. In the case of human beings, likewise, a dimeric-recessive determination has been suspected in the case of certain morbid conditions, but trustworthy proof of the hypothesis is still lacking. If one of two such genes were generally diffused throughout a population, then the other gene would show a simply recessive heredity. If both the heredity factors were comparatively rare, then among the sibs of the patient we should in an extreme case expect to find only 6·25 % (that is to say one sixteenth) of persons suffering from the same malady. In the case of heredity factors more frequently present, on the other hand, we should expect, notwithstanding the doubly recessive determination, to find a higher percentage of cases among the sibs, although this percentage could never reach 25. If, for instance, a patient has the formula *aabb*, he may be the child of two outwardly healthy parents having the formula *AaBb*. He would then have among others, healthy sibs of the formula *Aabb* or *aaBb*. In these cases the factor *A* would function as an inhibitory factor in relation to the morbific heredity factor *bb*; and the factor *B* would act as an inhibitory factor in relation to the morbific heredity factor *aa*. Many geneticists favour the hypothesis that such "inhibitory factors" exist, though no one has as yet succeeded in demonstrating their existence in connexion with particular diseases. At any rate we have a right to ask that such factors shall not be merely "assumed", but that the inferences to be drawn from such assumptions shall be worked out arithmetically and be then submitted to an unprejudiced comparison with the facts of experience. If the frequency of the character be n, and that of the two heredity factors x and y, then $x^2 \times y^2 = n$. For a given frequency of the character, the lower limit of the frequency of its appearance in the sibs of probands will be attained when x equals y. In this way, we can calculate the lower limit. The upper limit is 25 %.

When the heredity is dominant, the existence of "inhibitory factors" is often assumed to explain the frequently recorded skipping of one or several generations in the heredity. The theory is that, for instance, the manifestation of a morbific heredity factor *C* can be inhibited by a factor *D*, so that an individual having the formula *Ccdd* would be diseased, whereas an individual having the formula *CcDd* would be healthy. Then, in a population wherein the factor *D* did not exist, the factor *C* would manifest a simply dominant heredity; whereas if the factor *D* were widely diffused throughout the population, the heredity of the malady would be more or less frequently interrupted, so that the distinction of this dominant heredity from a recessive heredity would be difficult. There would, in fact, then be a certain likeness to recessive heredity. For, if the factor *C* were very generally diffused throughout the population, the presence or absence of the factor *D* would be decisive as to the presence or absence of the malady, and this malady would be simply recessive, seeing that patients would be *CCdd*. If both the factors were moderately frequent in a population, the malady would have a heredity in which dominance and recessiveness were combined, for its manifestation would depend upon the coincidence of a dominant factor with a recessive factor in the homozygotic state. But nothing of the sort has hitherto been positively demonstrated.

The converse assumption of "exciting factors" is likewise a favourite one. It is supposed that a dominant morbific heredity factor *E* is not sufficient unaided to induce a malady, which can only arise when the morbific heredity factor *F* is simultaneously at work. In a population wherein *F* was generally diffused, *E* would then manifest a simply dominant heredity; and, on the other hand, in a population in which E was generally present, the factor F would induce a simply dominant heredity of the malady concerned. Both the factors would be equally indispensable to induce the active manifestation of the disease; and which of the two factors was to be regarded as the "genuinely" morbific one and which as no more than the exciting factor, would then depend on the comparative frequency of their distribution throughout the population. An individual suffering from the disease would, in general, have the formula $EeFf$. We see, then, that the assumption of "exciting factors" as indispensable to the manifestation of dominant heredity factors coincides with the previously considered assumption of dimeric determination. Among the offspring of carriers of the character we must expect to find 25 % of carriers as a minimum—this minimum prevalence occurring when the character is a rare one and when the two heredity factors which determine it are both equally rare. But the more frequent one of the two heredity factors is, the nearer to 50 % will be the proportion of carriers of the character among the children of parents one of whom manifests the character. When one of the two determining heredity factors is widely diffused, the dimeric determination passes over into monomeric.

In general we shall do well to avoid assuming the probable working of these or other kinds of polymerism, until simpler possible explanations have been excluded. Investigators should be especially on their guard against laying too much weight upon the demonstration of theoretically accurate numerical ratios among the sibs. Until a few years ago, notwithstanding the difficulty of demonstrating accurate mendelian ratios in human beings, the demonstration of such ratios was still regarded as indispensable to accepting the validity of the mendelian law as far as human beings were concerned. Happily, as has already been said, this demand is no longer maintained in all its rigour. As far as the recognition of recessive heredity is concerned, the demonstration of theoretically correct ratios among the sibs is not a matter of primary importance. Here it is of more moment to ascertain the comparative frequency of consanguineous marriages among the parents. Above all in the case of heredity factors whose manifestations may in part depend upon the cooperation of environmental influences, the search for theoretically accurate mendelian ratios may actually lead us astray. Further, dominant factors which need the cooperation of external exciting causes in order to make them actively manifest will not be demonstrable in 50 % of the sibs, but only in a smaller proportion, which may fall as low as 25 %, thus simulating a recessive heredity. In such instances, we can have recourse (as an important method of distinguishing between dominant and recessive factors) to a comparison of the frequency of the character in the sibs with its frequency in the parents of the carriers of the character, or, it may be, also with its frequency in the children of the carriers. In the case of comparatively infrequent dominant heredity factors, their presence is to be expected in 50 % of the sibs and also in 50 % of the parents and 50 % of the children; and consequently their manifestation through the cooperation of environmental influences is likewise to be expected in an equally large percentage of sibs, parents, and children. Of course, age must be taken into account, and it would be best to restrict our comparison to groups of persons having the same age. In the case of not unduly frequent recessive heredity factors, on the other hand, we must expect to find them less often manifested in parents and in children of affected persons than in the sibs of these. When, for instance, a recessive factor whose frequency in a population is 1 %, manifests itself in only 5 % instead of in 25 % of the sibs of carriers of the character (because in the remaining 45 the exciting environmental influences have not been operative), then we must expect to find it in only 2 % instead of in 10 % of the parents and the children of the carriers of the character. In all cases where the carriers of a character have their reproductive capacity impaired, by the presence of the anomaly, the percentage of manifestations in the parents will lag behind the theoretical expectation, whereas the percentage of manifestations among the children will not thereby be affected. That is why Hoffmann, for instance, has with good reason made the children (rather than the parents) of persons suffering from mental disorder the object of special investigation. It is, of course, of great and direct value to ascertain the frequency of morbid conditions among the offspring of persons suffering from certain maladies. For practical purposes, such knowledge may be of more importance than a knowledge of the theoretical heredity.

Even if the heredity be of some other kind, we may expect, where there has been unselective mating, to find among the children of the carriers of a character just as many carriers of that character as we find among the parents of the probands, provided that the character has not been subjected to special selection. If the percentage among the children and parents is the same as the percentage among sibs, this also, in cases of polymeric determination, speaks in favour of dominance; but if the former percentage lags considerably behind the latter, that speaks in favour of recessiveness.

Rüdin has drawn attention to the fact that light upon the nature of the heredity may also be thrown by a comparison of the frequency of a character in the sibs and the half-sibs, respectively, of the carriers of a character. When the common parent of several half-sib series is a carrier of the character, the manifestation of the character in more than one half-sib series speaks strongly in favour of a dominant heredity. But even when the joint parent does not manifest the character (as may happen when the cooperation of external environmental influences is essential to the manifestation of the character), then we may expect to find, in the case of a dominant heredity, among the half-sibs on the average half as many carriers of the character as among the sibs proper; but in the case of a recessive heredity, we find that the ratio between carriers among the half-sibs and carriers among the sibs is much smaller than this, and that the expectation among half-sibs is only half as great as the expectation among the parents of the sibs. Of course we have to remember that even in a fairly large accumulation of data the number of half-sibs will be inconsiderable, so that the fallacy of small numbers will play a considerable part in our results.

A great difficulty in the discovery of numerical ratios arises from the circumstance that many hereditary maladies and other hereditary factors do not manifest themselves until the persons concerned are well on in years. This difficulty can only be overcome by drawing up for the malady under consideration a sickness table analogous to the familiar mortality tables—as Weinberg has shown in Rüdin's work on dementia praecox. The "compensation and exclusion method" utilised by J. Bauer and B. Aschner is only speciously precise, for the expectation of illness cannot be inferred from the ages of those who actually fall sick unless we have a knowledge of the general composition of the age classes.[1]

Of great importance when we are studying the heredity of conditions other than those which are beyond question monomerically determined is that we should attempt to form a clear picture of their *general frequency throughout the population.* In the case of recessive heredity factors the expectation of the frequency with which we shall find them manifest in the parents and in the children of carriers of the character, is mainly determined by the general frequency of the conditions throughout the population; and the rarer, in this respect, the particular factor is, the more frequently may we expect to find the extremely significant consanguinity of the parents. Reference has already been made to both these important matters. Trustworthy knowledge as to the frequency of morbid conditions will, indeed, only be rendered attainable by a general registration of the population. Extant medical statistics are inadequate for our purpose. All the same, we can get a vague sort of picture by random enquiries in sections of the population. Such inaccurate estimates of frequency as we are now able to make for certain conditions (hereditary deaf-mutism, insanity, etc.) are certainly better than no estimates at all—all the more, seeing that, for the elucidation of heredity, precision is often superfluous, it being enough to have a general idea whether the frequency of a malady approaches 1 : 10, 1 : 100, 1 : 1000, etc. If, for the origination of a malady, two distinct heredity factors are requisite, the rarer of these two is the one of more practical importance. I have already discussed the importance of such ratios of frequency[2] when giving an account of the various hypotheses which have been formulated regarding the hereditary determination of the schizophrenias.

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[1] Cf. W. Weinberg, Methoden und Technik der Statistik, a contribution to Gottstein's Handbuch der sozialen Hygiene, Springer, Berlin, 1925, vol. I, pp. 107–138.

[2] F. Lenz, Einige grundsätzliche Bemerkungen zur Fragestellung nach der erblichen Bedingtheit der Schizophrenien, "Archiv für Rassenbiologie", vol. XV, 1924, no. 3.

In the case of a sex-linked factor, the expected average frequency of its appearance in one sex can be calculated from its frequency in the other. If such a recessive sex-linked anomaly as red-green blindness is found in about 4 % of all men, it follows that approximately one in twenty-five of the sex chromosomes must contain the appropriate heredity factor, seeing that each man has only one sex chromosome. Conversely, in the female sex, where each individual has two sex chromosomes, such a heredity factor has a probability amounting to $\frac{1}{25} \times \frac{1}{25} = \frac{1}{625}$ of coinciding with another such heredity factor. In that case we should expect to find 0·16 % of women with the anomaly in question, unless the percentage were to be slightly increased by consanguineous marriages. If the frequency in the male sex be $\frac{1}{n}$, then in the female sex we may expect a frequency of $\frac{1}{n^2}$. The commoner a sex-linked recessive anomaly is, the more will its frequency in females approach its frequency in males. A sex-linked factor present in all males must also be expected in all females—regardless of the fact that it is sex-linked. In the case of sex-linked dominant factors, the frequency in females is of course greater than in males; but the differences in frequency in the two sexes are in general much smaller than in the case of recessive factors. If about one half of all men were to exhibit a sex-linked factor, it can easily be seen that we must expect to find this same factor in about three fourths of all women. If the frequency in the male sex be $\frac{1}{n}$, then in the female sex we may expect a frequency of $\frac{2}{n} - \frac{1}{n^2}$. In the case of very rare factors, we come to the limitary instances in which the frequency in females is twice as great as the frequency in males.

If, in any given case, the heretofore described simple possibilities of hereditary determination do not suffice to account for the facts, we must suspect that a more complicated polymerism is at work. As a rule when we are dealing with human material we shall find it impossible to elucidate polymeric determination fully. The elucidation of instances of dimeric determination may encounter insuperable obstacles—fortunately, however, we have no reason to believe that the problems of fundamental importance in practical racial hygiene are so complicated as this. The great majority of the numerous morbific heredity factors studied by Morgan in the fruit-fly and by Baur in the snapdragon could be classified either as dominant or as recessive, and there is no reason to suppose that matters are different as regards the morbific heredity factors in human beings. In the field of pure theory we must, no doubt, admit that strictly speaking every character is dependent upon the totality of all the genes (Weinberg speaks of this as "panmerism"); but practical experience speaks strongly for the view that most morbid trends are substantially dependent upon one or upon very few heredity factors, and that normal qualities, though usually dependent upon several heredity factors, are by no means dependent upon an unrestricted number. H. Federley[1] has, indeed, rightly insisted that, within the next few centuries, we cannot hope to be able to analyse successfully the more complicated instances of polymerism in human beings—and I myself believe that such problems will always prove beyond our powers to elucidate. Happily such extremely intricate cases cannot be of much moment in the study of hereditary diseases. I do not mean to imply that morbific heredity is never thus complicated, but in the majority of instances the problem is obviously much simpler.

Federley[2] experimented with various organisms, and notably with a butterfly, Spilosoma lubricipedum. In this insect, which ordinarily has whitish-yellow wings with black spots, there sometimes exists a heredity factor which produces an extension of the black-spotted areas. When homozygotically present, this factor may make the wings almost uniformly black, but in some of the homozygotes, owing to the constitution of the other heredity factors, we find no more than isolated black macules on the wings; and Federley tells us that specimens in which the factor is present heterozygotically may be so like the ordinary yellow form that distinction is difficult. We shall certainly find it impossible to clear up the heredity of analogous instances in human beings. I regard it as probable that analogous instances do occur in our own species. For instance, I can very well believe that something of the sort happens in the case of schizophrenia or of schizoid psychopathy.

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[1] Zur Methodik des Mendelismus in bezug auf den Menschen, "Acta Medica Scandinavica", vol. LVI, 1922.

[2] Die Bedeutung der polymeren Faktoren für die Zeichnung der Lepidopteren, "Hereditas", I, 1920.

In human beings we cannot hope to elucidate cases of *multiple allelomorphy* although they must be common enough (see above, p. 260). Only as regards what are spoken of as *blood groups* (isohaemagglutinins) does F. Bernstein appear to have succeeded in demonstrating multiple allelomorphy.[1] This, however, is an exceptional instance of allelomorphy in which the conditions are comparatively simple, inasmuch as there seem to be only two distinct isohaemagglutinins, and consequently no more than four blood groups (see above, pp. 163–4).

Nor will it as a rule be easy to demonstrate the *coupling of genes* in human beings, although, reasoning by analogy from what has been observed in animals and plants, we may confidently infer that such coupling occurs also in man. Now that, of late years, the study of the coupling of genes has been so fruitful in experimental heredity, a number of medical writers have expressed the view that geneticists ought to make similar investigations in the domain of human heredity. Most of these writers have failed to draw a clear distinction between coupling and correlation, J. Bauer being especially confused upon this point. They have overlooked the fact that a gene which is coupled with another gene, is coupled to the same extent with all the genes allelomorphic to that gene. But genes which are allelomorphic one to another tend in part to determine the production of contrasted characters. The result is, therefore, that the coupling of genes does not produce any correlation of qualities within a mixed population, but only within a sib series. Within a sib series, however, we cannot demonstrate coupling satisfactorily, owing to the fallacy of small numbers. In the case of the fruit-fly Drosophila, we can breed hundreds of sib series, so that the demonstration of coupling is easy enough; but in human beings it is practically impossible.

Furthermore, the coupling of genes is to be expected much less frequently in human beings than in Drosophila. Seeing that in Drosophila there are only four pairs of chromosomes, we must expect with a high degree of probability that a particular gene will have coupled relations with approximately one in four of the other genes. On the other hand in human beings (who have apparently 24 pairs of chromosomes), a gene will on the average be coupled with 1 in 24 of the other genes. Only in the case of sex-linked heredity factors are we entitled to assume that, since they all lie in the same chromosome, they will all be coupled one with another; but even then we must suppose that the coupling is not absolute but merely relative.[1]

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[1] Zusammenfassende Betrachtungen über die erblichen Blutstrukturen des Menschen, "Zeitschr. f. induktive Abstammungs- und Vererbungslehre", vol. XXXVII, 1925, p. 237.

In Drosophila an exchange of genes between allelomorphic chromosomes occurs only in the female sex. We do not know whether the same thing applies to human beings, and we can hardly expect the point to be cleared up for some time to come. This must be borne in mind when formulating hypotheses with regard to the coupling of genes in the human species.

The demonstration of a *correlation* of characters is of great importance for the solution of the problem whether two or more conditions may be wholly or partly determined by one and the same heredity factor. M. Pfaundler has studied this matter clinically.[2] We speak of two conditions as correlated when they occur conjoined more frequently than should be expected from their frequency in isolation.[3] When two conditions are encountered invariably conjoined, their correlation is complete (= + 1); when two conditions are met with conjoined less often than we should expect in view of their frequency in isolation, the correlation is said to be negative; in the extreme case when both conditions are mutually exclusive, the correlation = − 1. Correlation is non-existent when the conditions appear conjoined neither more frequently nor less frequently than should be expected from their frequency in isolation—when, that is to say, the frequency of conjunction is exactly equal to the product of the separate frequencies (or, which comes to the same thing, the product of the separate probabilities). The degree of correlation is measured by the extent to which the probability of the conjunction of two conditions differs from the product of the isolated probabilities.

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[1] F. Lenz, Koppelung mit dem Geschlecht oder Lokalisation im Geschlechtschromosome?, "Zeitschrift f. induktive Abstammungslehre", vol. XXVIII, 1922, p. 243.

[2] M. Pfaundler and L. Seht, Ueber Syntropie von Krankheitszustände. "Zeitschr. f. Kinderheilkunde", 1921.

[3] The notion of correlation can be equally well applied to series composed of more than two units. Here, however, it will suffice to consider the simplest case.

Its calculation may best be illustrated by an example. In material studied by Pfaundler, among 34,555 children, 764 suffered from goitre, 87 from cretinism, and 80 from both conditions combined. From these data we may construct the following table of correlation:

	Cretinism	No cretinism
goitre	80	684
no goitre	7	33,784

The correlation coefficient (according to Bravais and Pearson) is

$$r = \frac{ad - bc}{\sqrt{(a + b)\,(c + d)\,(a + c)\,(b + d)}}$$

where the letters represent as follows the figures in the correlation table:

a	b
c	d

a thus denotes the number of children with both conditions (80), *b* the number of children with either goitre or cretinism (684), *c* the number of children with cretinism but no goitre (7), *d* the number of children with goitre but without cretinism (33,784). The sum total of the children is $a + b + c + d = 34{,}555$; that of the children with goitre, $a + b = 764$; that of the children with cretinism, $a + c = 87$; that of the children free from goitre, $c + d = 33{,}791$; that of the children free from cretinism, $b + d = 34{,}468$. In this case, then, the correlation coefficient is

$$r = \frac{80 \times 33{,}784 - 684 \times 7}{\sqrt{764 \times 33{,}791 \times 87 \times 34{,}468}} = + 0{\cdot}31$$

In the case of correlation coefficients, the mean error of small numbers is expressed by the value $\frac{1 - r^2}{\sqrt{n}}$. The calculation gives 0·005. The correlation between goitre and cretinism is therefore $r = + 0{\cdot}31 \pm 0{\cdot}005$. Pearson's coefficient, consequently, does not in this case yield a very strong correlation, although the coupling between cretinism and goitre is so strong that of 87 cretinoid children only 7 are free from goitre. A stronger coupling is shown by my own "correlation index":

$$k = \tfrac{1}{4}\left(\frac{a-c}{a+c} + \frac{d-b}{d+b} + \frac{a-b}{a+b} + \frac{d-c}{d+c}\right)$$

This yields the value $k = 0{\cdot}50 \pm 0{\cdot}004$.

A much higher value is given by Yule's association coefficient, $ass = \dfrac{ad - bc}{ad + bc} = 0{\cdot}98$, but this coefficient is invalidated by a theoretical weakness, for, should either of the groups *b* or *c* not be represented, it always yields + 1 and, conformably, should either of the other groups *a* or *d* be unrepresented, it always yields − 1. It should be obvious, however, that, for instance, the correlation between goitre and cretinism must not be considered absolute even were no cases of cretinism without goitre forthcoming, for there would still be forthcoming very numerous cases of goitre without cretinism, and only if no such cases existed would the correlation be absolute, for then only would both the correlation coefficient and the correlation index be equal to + 1.

Pearson, too, has found that the correlation coefficient often gives too small a value in the case of a relationship between two pairs of alternatives, and he therefore gives a special four-fields coefficient, which however not only involves extremely elaborate calculations, but is questionable as to its foundations. For my part, therefore, I prefer the correlation index, which in the case of alternative reciprocal relationships is usually higher than the correlation coefficient, and consequently gives better expression to the actual connexion.

Often, too, the correlation calculus is directly applied to the elucidation of heredity. In this case, it serves as a measure of the average resemblance between relatives of some particular degree of nearness, as, for instance, between parents and children.

The use of the calculus for this purpose may be illustrated by an imaginary example. Let us suppose that a character is present in 1 % of the population. It will then manifest itself in 100 among 10,000 fathers subjected to examination; and among 100 of the firstborn children of the 100 fathers, 10 will exhibit the character; whilst, in general, in the same generation of children, the character will be present in the ratio of 1 : 100, and will therefore be found in 100 of the firstborn children of all the 10,000 fathers. We can, therefore, draft the following correlation table to show the family grouping in respect of the anomaly, + indicating that the anomaly is present and — indicating that it is absent:

	child +	child —
father + ...	10	90
father —	90	9810

We get a correlation index[1] + 0·09, this apparently denoting a weak "hereditability" of the character. Nevertheless, the character may be, for practical purposes, completely independent of external influences, and therefore wholly determined by heredity. For, indeed, a simply recessive factor occurring in the population with a frequency of 1 : 100 would recur in the offspring of carriers of the character in the same proportion as is shown in the correlation table (see above, pp. 521 et seq.). We see from this that the correlation calculus is a very defective measure of the heredity.

Certainly it gives us some indication of the heredity. If heredity played no part in the matter—if, in other words, the occurrence of the anomaly in both fathers and children were solely due to chance—the distribution would be as follows:

	child +	child —
father + ...	1	99
father —	99	9801

In that case the correlation = 0. If, however, the actual heredity be often thus indicated in the correlation, we must never forget that we have to do with an equation containing several unknown quantities. Apart from environmental influences, the course of the heredity and the frequency of an anomaly affect the correlation. Let us assume that a recessive factor is present in one fourth of the population. In that case, as we can easily calculate, we should expect the anomaly to occur in half of the offspring of carriers of the character. In 10,000 family groups, we should get the following distribution:

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[1] When $b = c$, when that is to say we have a symmetrical distribution of the correlation table, we get identical values for the correlation index and the correlation coefficient.

	child +	child −
father + ...	1250	1250
father −	1250	6250

As correlation we get here 0·33 (precisely $\frac{1}{3}$), this being a much higher value than if the character were rarer—although the course of the heredity is unchanged. All the same, despite the untrustworthiness of the correlation calculus, we cannot completely dispense with it in genetics.

The correlation calculus is, in genetics, especially applied to characters such as do not enable us to classify the sibs in sharply distinguished groups—to characters taking the form of quantitative differences with numberless intermediate gradations. (That is what we generally find in normal characters, such as stature, in contradistinction to morbid characters.)

We may take stature as an example to show the form assumed in such instances by the correlation calculus. Here we can only construct artificial groups, placing in one group, for instance, all the individuals whose stature is denoted by the same number of integral centimetres. If that basis of classification be regarded as unduly detailed, we range the groups according as the individuals in each group differ from those of the next group by two centimetres, or by five centimetres, or by some other number of centimetres as the case may be. When we wish to compare parents with children we must further ascertain how the children of respective statures are distributed among the various parental groups. We can then arrange our data in a correlation table as seen on the opposite page.

The figures in the correlation table give the number of families in which a particular stature of the parent coincides with a particular stature of the children. The investigation concerned 928 families. In such cases the stature of the female is converted into the corresponding male stature in accordance with the usual ratio between male and female; then a mean is taken between the stature of the two parents and a mean is also taken of the stature of the children of each pair. If, however, we wish only to ascertain the correlation between fathers and sons, or that between fathers and daughters, etc., we do not effect these conversions.

From this correlation table we can recognise at the first glance that, on the average, the stature of the children increases proportionally with the stature of the parents, so that there is a positive correlation between the two. If there were complete correlation (= + 1), all the figures would be concentrated into the diagonal running from the left upper corner to the right lower corner; and if there were complete negative correlation, they would be concentrated into the diagonal running from the right upper corner to the left lower corner.

		Stature of the Parents (Series X)											Number of cases	Average of parents	Aggregate average of parents
		63·5	64·5	65·5	66·5	67·5	68·5	69·5	70·5	71·5	72·5	73·5			
Stature of the Children (Series Y)	61·2	1	1	1	—	—	1	—	1	—	—	—	5	66·4	
	62·2	—	1	—	3	3	—	—	—	—	—	—	7	66·6	
	63·2	2	4	9	3	5	7	1	1	—	—	—	32	66·3	
	64·2	4	4	5	5	14	11	16	—	—	—	—	59	67·8	
	65·2	1	1	7	2	15	16	4	1	1	—	—	48	67·9	
	66·2	2	5	11	17	36	25	17	1	3	—	—	117	67·7	
	67·2	2	5	11	17	38	31	27	3	4	—	—	138	67·9	68·303
	68·2	1	—	7	14	28	34	20	12	3	1	—	120	68·3	
	69·2	1	2	7	13	38	48	33	18	5	2	—	167	69·5	
	70·2	—	—	5	4	19	21	25	14	10	1	—	99	69·0	
	71·2	—	—	2	—	11	18	20	7	4	2	—	64	69·0	
	72·2	—	—	1	—	4	4	11	4	9	7	1	41	70·0	
	73·2	—	—	—	—	—	3	4	3	2	2	3	17	70·4	
	74·2	—	—	—	—	—	—	5	3	2	4	—	14	70·9	
Number of cases		14	23	66	78	211	219	183	68	43	19	4	928	—	
Average of children		65·3	65·8	66·7	67·2	67·6	68·2	68·9	69·5	69·9	72·2	72·7	—	—	
Aggregate average of children							68·095								

Correlation between the stature of parents and the stature of their adult children. (Based upon Galton's data. Measurements in inches.)

As a measure of the correlation, we may use the Bravais correlation coefficient as introduced by Karl Pearson.[1] Its nature will be most readily understood from a study of what is termed *regression.* In this material relating to stature, we see that the children do not diverge from the mean so much as their parents do, the divergence being only about two thirds as great. We say, therefore, that their "regression" towards the parents amounts to 0·68.

If the parents are arranged in classes according to stature, the average stature of the children of the parents in the respective classes is as follows:

Parents	63·5	64·5	65·5	66·5	67·5	68·5	69·5
	70·5	71·5	72·5	73·5			
Children	65·3	65·8	66·7	67·2	67·6	68·2	68·9
	69·5	69·9	72·2	72·7			

The average stature of the parents was 68·364 in., that of the children 68·086 in. Thus for the ratios between the deviations from the average we get in the various classes the following regression figures:

0·57 0·59 0·48 0·48 0·56 0·84 0·72 0·66 0·58 0·99 0·90

The number of families in the various classes was as follows:

14 23 66 78 211 219 183 68 43 19 4

From these figures we deduce a mean regression of 0·68.

"Regression", therefore, does not signify (as is often erroneously assumed) a "reversion" towards the mean, but the very opposite of this. It may be regarded as a measure of the average heredity, as a "heredity gauge". The dependence of the stature of children upon the stature of parents is shown by Fig. 160. The irregularities in the curve are due to the error of small numbers; were this error eliminated, we should get a mean line of regression.

Conversely, we may range the children in classes according to their stature, and then ascertain what is the relation of the stature of the parents to that of the children (see Fig. 161).

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[1] The mathematician Bravais did not originate the formula for the measurement of correlation, but for a study of error in physical observations. Pearson subsequently applied it to the measurement of the mutual relationships between two series of observations, that is to say, two correlations.

This figure is constructed from the same material as Fig. 160. It shows that there is also a regression of the parents towards the children, amounting, however, on the average, to only

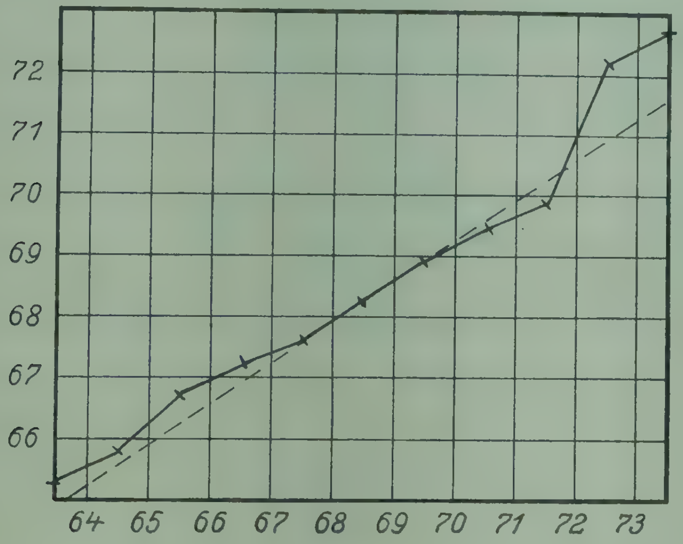

FIG. 160.
The regression of the stature of children towards that of the parents (based on Galton's data).

0·33 per cent. At first this seems very remarkable, for, inasmuch as the parents naturally have as large a proportion of their hereditary equipment in common with the children as

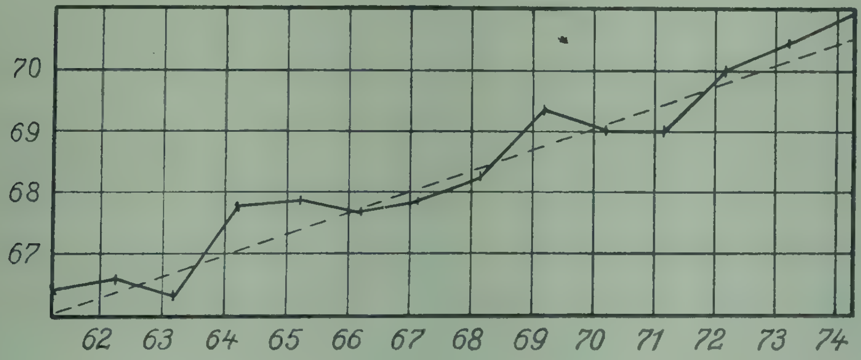

FIG. 161.
The regression of the stature of the parents towards that of the children (based upon Galton's data).

the children have in common with the parents, we should expect to find that the "heredity gauge" would be the same in both instances. The reason for the apparent deviation is that in the parental generation we are dealing with a mean of the stature of the two parents, whereas the stature of the children represents individual data. Now, the variability of the mean of the parental pairs is little more than half the variability of the parental generation; and the regressions are dependent on the ratio between the variabilities. If the variability of the children were the same as that of the parents, the two regressions would be identical, and obviously in that case the figure would be the given "heredity gauge". Since, however, in reality the regressions seldom coincide, we take as measure of the heredity the geometrical mean (that is to say the square root of the product) of the regressions as our heredity gauge. This is the correlation coefficient.

The regression of series Y towards series X (in our case that of the children towards the parents) is $R\frac{y}{x} = \frac{\Sigma p a_x a_y}{n\sigma_y^2}$, and the regression of series X towards series Y (in our case the regression of the parents towards the children) is $R\frac{x}{y} = \frac{\Sigma p a_x a_y}{n\sigma_x^2}$. The geometrical mean of the two regressions is the correlation coefficient $r = \frac{\Sigma p a_x a_y}{n\sigma_x\sigma_y}$.

The significance of the algebraical signs can be best explained with reference to our concrete example. a_x denotes the deviation of the stature of the parents of a family group from the average of the parents, a_y denotes the deviation of the stature of the children of a family group from the average of the children. $a_x a_y$ is, therefore, the product of these two deviations of a family group; p denotes the number of families in each group, and $\Sigma p a_x a_y$ denotes the sum of the deviation products of all the family groups; n denotes the total number of the families, which is here 928; σ_x is the square root of the arithmetical mean of the squares of the parental deviations from the average of the parents, and σ_y is correspondingly the square root of the arithmetical mean of the squares of the children's deviations from the average of the children; consequently, $\sigma_x = \sqrt{\frac{\Sigma a_x^2}{n}}$, and $\sigma_y = \sqrt{\frac{\Sigma a_y^2}{n}}$.

If we wanted to calculate the correlation coefficients directly in accordance with the formula $r = \frac{\Sigma p a_x a_y}{n\sigma_x\sigma_y}$, the calculation would be extremely particularised and tedious, for the deviations from the average are very rarely integral numbers, and to obtain their products and squares we should have to deal with many places of decimals. Fortunately, however, there is a much simpler way of calculating *r*, one in which integrals can be mainly employed. I will illustrate this method briefly from our example.[1]

Sufficient precision is secured by ranging our material in accordance with gradations of stature amounting to two inches instead of only one. We then get the following table:

	Stature of the Parents (Series X)						Number of Cases	
Stature of the Children (Series Y)	64	66	68	70	72	74		
60·7	2	1	1	1	—	—	5	
62·7	7	15	15	2	—	—	39	$e_y = 2 \cdot 075$
64·7	10	19	56	21	1	—	107	
66·7	14	56	130	48	7	—	255	$M_y = 68 \cdot 086$
68·7	4	41	148	83	11	—	287	
70·7	—	11	69	66	17	—	163	$\sigma_y = 2 \cdot 583$
72·7	—	1	11	22	20	4	58	
74·7	—	—	—	8	6	—	14	
Number of cases	37	144	430	251	62	4	928	
	$e_x = 1 \cdot 419$ $M_x = 68 \cdot 364$ $\sigma_x = 1 \cdot 835$							

Instead of setting out from precise average values, we set out from the classes A_x and A_y, which adjoin the average values. We choose the X class 68 and the Y class 68·7 as starting-points. The divergence of class A_x from the mean M_x is $b_x = M_x - A_x$; the divergence of class A_y from the mean M_y is $b_y = M_y - A_y$.

[1] The reader who wants fuller details concerning these methods of calculation should consult W. Johannsen, Elemente der exakten Erblichkeitslehre, third edition, Fischer, Jena, 1926.

$b_x = 68{\cdot}364 - 68{\cdot}000 = \quad 0{\cdot}364 = \quad 0{\cdot}182$ of a 2 in. gradation
$b_y = 68{\cdot}086 - 68{\cdot}700 = -0{\cdot}614 = -0{\cdot}314$ of a 2 in. gradation

We can now obtain the correlation coefficient r by means of the formula:

$$r = \frac{\Sigma p a_x a_y - n b_x b_y}{n \sigma_x \sigma_y}$$

σt is obtained by means of the formula:

$$\sigma_x = \sqrt{\frac{\Sigma p a_x^2}{n} - \sigma_x^2}$$

The 144 pairs of parents in the first x class to the left of the class taken as starting-point deviate from this class by the extent of one gradation. A_x^2 is therefore equal to 1. The 37 pairs of parents in the second class to the left of the class taken as starting-point deviate from this class by the extent of two gradations, and here, therefore, $a_x^2 = 4$; and so on.

$$\Sigma p a_x^2 = \begin{array}{r} 144 \times 1 = 144 \\ 251 \times 1 = 251 \\ \hline +395 \end{array} \quad \begin{array}{r} 37 \times 4 = 148 \\ 62 \times 4 = 248 \\ \hline +396 \end{array} \quad \begin{array}{r} 4 \times 9 = 36 \\ \\ \hline +36 \end{array} = 827$$

$$\begin{array}{r} 827 : 928 = 0{\cdot}891 \\ b_x^2 = 0{\cdot}033 \\ \hline \text{difference} = 0{\cdot}858 \end{array}$$

$\sigma_x = 0{\cdot}926$ gradation $= 1{\cdot}853$ in.

Correspondingly therefore $\sigma_y = 1{\cdot}291$ gradation $= 2{\cdot}583$ in.

Now we have to construct the products $a_x a_y$. In the areas corresponding to the classes taken as starting-points, the products $= 0$, for there, either a_x or $a_y = 0$. In the left upper squares: for the one containing the number 56, $a_x = 1$ and $a_y = 1$, so that $a_x a_y = 1$ and $p a_x a_y = 56$; for the square containing the number 19, $a_x = 1$ and $a_y = 2$, so that $a_x a_y = 2$, and $p a_x a_y = 38$; and so on. In the second and third squares the products are negative, for in the second square a_x is negative, and in the third square a_y. In all, we get the following products:

Square I	Square IV	Square II	Square III
$56 \times 1 = 56$	$66 \times 1 = 66$	$48 \times 1 = 48$	$11 \times 1 = 11$
$14 \times 2 = 28$	$17 \times 2 = 34$	$7 \times 2 = 14$	$1 \times 2 = 2$
$19 \times 2 = 38$	$22 \times 2 = 44$	$21 \times 2 = 42$	-13
$10 \times 4 = 40$	$20 \times 4 = 80$	$1 \times 4 = 4$	
$15 \times 3 = 45$	$4 \times 6 = 24$	$2 \times 3 = 6$	
$7 \times 6 = 42$	$8 \times 3 = 24$	$1 \times 4 = 4$	
$1 \times 4 = 4$	$6 \times 6 = 36$	-118	
$2 \times 8 = 16$	$+308$		
$+269$			

Sum of the positive products = 577
,, ,, negative ,, = 131

$$\Sigma p a_x a_y = 446$$

$$nb_xb_y = 928 \times 0\cdot182(-0\cdot314) = -\ 53\cdot032$$
$$\Sigma p a_x a_y - nb_xb_y = 499\cdot032$$

$$n\sigma_x\sigma_y = 1109\cdot7$$
$$r = 499 : 1110 = 0\cdot45$$

According to Pearson and Filon, the mean error of the correlation coefficients is to be calculated by the formula $\frac{1 - r^2}{\sqrt{n}}$, and is found to be 0·026.

Consequently **$r = + 0\cdot45 \pm 0\cdot026$.**

There is no object in calculating a correlation coefficient to beyond two places of decimals. If we do so, we are merely simulating an accuracy which is, in the circumstances, unattainable.

The *correlation index* (whose use was suggested by myself) is based, not upon the products of the deviations, but upon the deviations themselves; and it is brought into relation, not with the mean quadratic deviation σ, but with the mean deviation e. Its formula is

$$k = \frac{1}{2}\left(\frac{S_x\,\text{I, IV} - S_x\,\text{II, III}}{S_x} + \frac{S_y\,\text{I, IV} - S_y\,\text{II, III}}{S_y}\right)$$

or $$k = \frac{1}{2n}\left(\frac{S_x\,\text{I, IV} - S_x\,\text{II, III}}{e_x} + \frac{S_y\,\text{I, IV} - S_y\,\text{II, III}}{e_y}\right)$$

Here the correlation table is regarded as divided into four equal quadrilateral fields by the lines of the mean values in the X direction and the Y direction. S_xI is the sum of the X deviations in the first field, S_xII in the second field, and so on. S_x is the absolute (non-algebraical) sum of all the X deviations. In the values + 1 and − 1, the correlation index when the regression runs in a straight line coincides with the correlation coefficient. In the values lying between these extremes, certain differences result from the fact that in the calculation of the correlation coefficient the extreme deviations raised to a higher power have to be taken into account, whereas in the calculation of the correlation index they have to be taken into account only proportionally to their magnitudes not thus raised. Here the correlation index seems to me to have an advantage theoretically as well as practically. Usually the index is somewhat larger than the coefficient, the index being the arithmetical mean between two fractions, whilst the coefficient is a geometrical mean. The difference reaches its maximum in the case of alternative variability, where (as is well known) the values given by the correlation coefficient are usually too small. The four fields coefficient, recommended by Karl Pearson for alternative variability, does indeed yield higher values, but has a dubious theoretical foundation and is in practice hard to calculate.

The calculation of the correlation index is, as a rule, much simpler than that of the correlation coefficient; and it can for the most part be effected with integral figures. In my contribution to E. Gotschlich's work on the methods of hygienic research, I have discussed its calculation as regards Galton's data concerning stature.[1] To this I must refer the reader, for considerations of space make it impossible to deal with the matter here.

If with the aid of the calculus of correlation an investigator wishes to gain information as to how far, in a given population, a character depends upon hereditary predisposition and how far upon environmental influences, he will do well to form a clear idea to what extent correlations are to be expected in the case of characters exclusively determined by the hereditary equipment. Let us suppose that a population has an F_2 composition, and that marriages occur without reference to the character with which we are concerned. In case of alternative variability, we should expect between fathers and sons a correlation of $+ 0 \cdot 33$ $(= \frac{1}{3})$, as we can see from the example given on pp. 539–540. In case of an intermediate relationship, the correlation would be $+ 0 \cdot 5$. The correlation between sibs would be $0 \cdot 42$ if the factor were dominant-recessive, and $0 \cdot 5$ if it were intermediate.

Now let us compare with this calculus certain correlations found by actual observation. Pearson and his collaborators have calculated quite a number of correlations between parents and children, on the one hand, and sibs, on the other. I will give here the correlations for colour of the eyes, since this character is practically independent of environmental influences:

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[1] Handbuch der hygienischen Untersuchungsmethoden, Methoden der menschlichen Erblichkeitsforschung, Fischer, Jena, 1929.

father and son	0·55	brother and brother	0·52
father and daughter	0·44	sister and sister	0·45
mother and son	0·48	brother and sister	0·46
mother and daughter	0·51		

The trifling differences between these figures are probably to be accounted for in the main by the error of small numbers. Apart from this, the frequency of characters in a population has an important influence upon the correlation coefficient. When a dominant character is comparatively rare, we may expect an increase in the correlation (see above, pp. 537 et seq.). Thus in Sweden, where a dark tint of the eyes is rarer than in England, we should expect to find a higher correlation between parents and children in respect of the colour of the eyes. We see, then, that we must not because of a low correlation value infer without more ado that a character is only to a small extent hereditary.

When in the case of the colour of the eyes the correlation is about 0·5, this must certainly not be taken as meaning that the colour of the eyes is about half determined by hereditary predisposition and about half determined by environmental influences. The dependence of the correlation upon the frequency of the heredity factors in the case of dominant-recessive behaviour with alternative variability is manifest in the following table:

Frequency of the dominant hereditary factor	0·0	0·1	0·2	0·3	0·4	0·5	0·6	0·7	0·8	0·9	1·0
Frequency of the recessive hereditary factor	1·0	0·9	0·8	0·7	0·6	0·5	0·4	0·3	0·2	0·1	0·0
Correlation between parents and children	0·50	0·47	0·44	0·41	0·38	0·33	0·29	0·23	0·17	0·09	0·00

Here it is assumed that the coincidence of the factors occurs exclusively in accordance with the measure of the frequencies, and that external influences play no part whatever in the manifestation of the trend. In the case of intermediate behaviour, we must always expect a correlation of 0·5 between parents and children, no matter the frequency with which intermediate factors are distributed; and the same applies as between sibs. The course of the curves is shown in Fig. 162.

If the frequency of the recessive heredity factor be m, then, according to W. Weinberg,[1] the correlation between parents and children is $\frac{m}{1+m}$ and between sibs $\frac{1}{2}\left(\frac{m}{1+m}+\frac{1}{2}\right)$.

When the manifestation of a heredity factor is inhibited by environmental influences, the correlation will be lower in proportion to the greater frequency of this inhibition. The following series of figures indicates the course of the correlation between parents and children as the frequency of the inhibition by environmental influences increases:

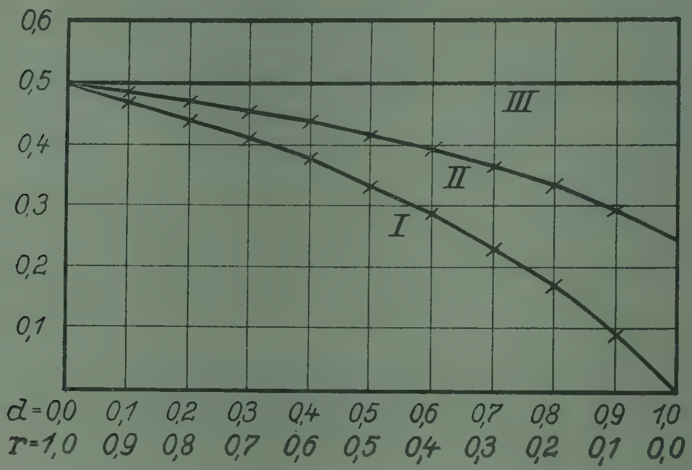

FIG. 162.

Correlation for varying frequencies of alternative heredity factors.

Curve I. Correlation between parents and children for dominant behaviour.
Curve II. Correlation between sibs for dominant behaviour.
Curve III. Correlation for intermediate behaviour.

The dominant factor is suppressed in	0·0	0·1	0·2	0·3	0·4	0·5	0·6	0·7	0·8	0·9	1·0
Correlation between parents and children	0·33	0·23	0·17	0·12	0·09	0·07	0·05	0·03	0·02	0·01	0·00

Here it is assumed that the frequency of the dominant factor is 0·5, and therefore that the frequency of the recessive factor is the same. The correlation between sibs declines from 0·42 to 0. For intermediate behaviour, the correlation both between parents and children and between sibs declines from 0·5 to 0. Fig. 163 shows the form of these curves.

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[1] Vererbungsgesetze beim Menschen, "Zeitschrift für induktive Abstammungs- und Vererbungslehre", vols. I and II, 1908 and 1909.

The correlation (which even if the character be exclusively determined by hereditary influences is not to be expected to be as high as 1) thus declines rapidly as the effect of environmental influences increases. There are certain ways, however, by which the correlation between relatives can be increased by environmental influences, as, for instance, when blood relatives, living together, are continually exposed to the same influences.

An additional circumstance whereby the correlation between blood relatives can be increased is the choice of persons of some particular type as sexual partners ("assortative mating").

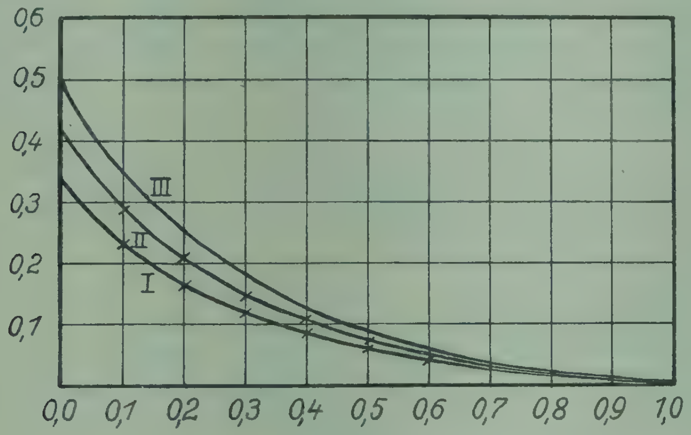

FIG. 163.

Correlation when a heredity factor having a frequency of 0·5 has its manifestation increasingly suppressed by environmental influences.

Curve I. Correlation between parents and children for dominant behaviour.
Curve II. Correlation between sibs for dominant behaviour.
Curve III. Correlation for intermediate behaviour.

When, for instance, European families are dwelling in tropical countries amid a coloured population, without mixing with that population to any notable extent, the correlation between parents and children will naturally be a very high one. For these reasons, correlation coefficients are useful as indications of heredity only within a particular population and in a particular environment. The same remark applies to the concept of resemblance in general. Ten individuals taken at random from among our population are, in general, extremely unlike any other ten individuals taken at random. But if two such groups of ten Germans or ten Englishmen were living among a Negro population, the resemblances between the respective groups would be far more striking than the differences. It is not improbable that the comparatively high correlations which have been found for stature and for the tint of eyes are partly dependent upon this preference for like persons in marriage. It is a matter of everyday experience that very tall persons are unlikely to marry very short ones; and, as Karl Pearson has shown, the same thing applies (though to a lesser degree) to the tint of the eyes.

Thus the correlation between blood relatives is dependent upon a number of influences; not only upon the relationship between heredity factors and the environment, but also upon the number of the genes concerned, upon whether their behaviour is dominant or intermediate, upon their frequency, and ultimately also upon assortative mating. If, therefore, we find the correlation between parents and children in respect of stature to be 0·45, this figure can by no means be regarded as giving us an unambiguous indication of the heredity.

C. B. Davenport,[1] in his investigations concerning the inheritance of stature, has divided the persons under study into three classes, tall, medium, and short (or else into five classes, the very tall and the very short being put into separate classes). The various combinations among the parents (when there is a three-class classification there are six combinations) were then separately considered. The investigation showed that out of a union tall × tall there proceeded children which on the average were somewhat taller than the parents, thus diverging a little farther from the mean than did the parents. In other words, in this group the regression exceeded 1. The offspring of a union small × small, on the other hand, diverged less from the mean than did the parents, so that here the regression was less than 1. I do not think, however, that we are justified in inferring from these results that the tendency to great stature is predominantly determined by recessive heredity factors and the tendency to small stature predominantly determined by dominant heredity factors. Since the offspring were in general taller than the parents, we have good reason to suppose that there has actually been a general increase in stature during recent generations, as the outcome of environmental influences (better feeding, etc.). Here we have another indication of the difficulties attendant upon the analysis of the heredity of normal qualities, which, as already pointed out, have, as a rule, a markedly polymeric heredity; and these difficulties are intensified by the collaboration of environmental influences. It is, on the other hand, much easier to trace the heredity of morbid deviations of stature, as we learn from a study of dwarfs (see above, pp. 317 et seq.).

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[1] Inheritance of Stature, "Eugenics Record Office Bulletin", 1917, no. 18.

For practical purposes, fortunately, the precise study of polymeric determination is of no great importance. The larger the number of the heredity factors that combine to produce a hereditary quality, that is to say the more polymeric such a quality is, the less practical moment has any single one of these heredity factors, and (for equal frequencies of the quality) the commoner throughout the population must be each of the collaborating genes. If, on the other hand, the frequency of the collaborating genes throughout the population varies, then the rarer genes are those of most practical importance. If, for instance, a morbid state is determined by several genes, of which only one is rare, this one has to be regarded as the genuinely morbific factor (see above, p. 533). Similar considerations apply to the respective importance of heredity factors and of environmental influences in the production of disease. If, for instance, a heredity factor, though widely diffused, only becomes manifest when made active in very exceptional instances owing to the collaboration of some particular environmental influence, this environmental influence is of more practical importance than the heredity factor. If, on the other hand, the exciting environmental cause is very widely diffused, whereas the heredity factor is rare, then this latter is for practical purposes the more important cause.

More fruitful than attempts at the mendelian analysis of polymeric qualities is, in most cases, the discovery of the frequency with which the carriers of particular characters are found among persons of near kin. In the present work, during the description of the hereditary diseases, and especially of the insanities, such frequencies were stated whenever possible. Always in these cases, we must enquire how large a percentage of the sibs, the parents, and the children of the carriers of a character are themselves also carriers of that character; and we must study the constitutions of the relatives in other respects. How large a proportion of the carriers of the character are the children of parents who display the same character?

How do the numerical ratios among the offspring vary when both parents are affected, or only one of the two parents is affected, or neither of the two affected? V. Haecker and T. Ziehen[1] have used this method of percentage calculation with excellent results in the study of the heredity of musical talent. They divided the parents of persons with marked musical talent into various groups, according as both parents were notably musical, notably unmusical, or one musical and the other unmusical; within the last group, again, a distinction was made between marriages in which the father was musical and those in which the mother was musical, since this point is of importance in view of a possible sex-linking of the character. The same method can of course be applied to all other qualities, the number of groups having to be used in each case for the purposes of classification varying in accordance with the nature of the material.

The *study of twins* is of especially great value as providing information concerning the respective importance of hereditary predispositions and environmental influences. Qualities which are exclusively or almost exclusively determined by the hereditary equipment appear in like manner and to the same degree in both members of a pair of mon-oval twins, this applying to polymeric as well as to monomeric qualities. That is why mon-oval twins are often so much alike that even their habitual associates find it difficult to tell them apart. No doubt mon-oval twins are usually exposed to almost identical environmental conditions—but the same thing applies to di-oval twins. It follows that a comparison of the results of observations on mon-oval twins with the results of observations on di-oval twins will be of the utmost value in enabling us to ascertain the relative importance of heredity and environment in producing certain qualities. By a flash of genius, Francis Galton recognised this a long while ago.[2] H. Poll[3] was one of the first in Germany to follow Galton's lead; and of late years the study of twins has been a favourite branch of genetic research.

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[1] Zur Vererbung und Entwicklung der musikalischen Begabung, Barth, Leipzig, 1923.

[2] The History of Twins as a Criterion of the Relative Powers of Nature and Nurture, "Journal of the Anthropological Institute", 1876.

[3] Ueber Zwillingsforschung als Hilfsmittel menschlicher Erbkunde, "Zeitschrift für Ethnologie", vol. XLVI, 1914.

Mon-oval twins regularly proceed from the same hereditary equipment. Exceptions to this rule, which might arise from a disturbance of the chromosome distribution, are, indeed, conceivable, but must be no less rare than are such disturbances in general. H. W. Siemens[1] has erroneously supposed that the first division of the fertilised ovum (a division which, when it goes too far, leads to the production of mon-oval twins) is a reduction division. This is an error; the first division of the fertilised ovum is an ordinary cell division. Observations made on klons and pure lines, that is to say, on organisms which reproduce themselves for many generations without fertilisation while retaining their hereditary qualities unimpaired, show that the ordinary cell divisions which occur in the course of development are divisions in which the hereditary equipment is equally distributed. Mon-oval twins in the human species are, in fact, analogous to a pure line; and just as in the pure lines of plants we can make a special study of non-inheritable environmental effects, so by the study of mon-oval twins in the human species we can learn to what extent a character can be modified by environmental influences.

We find, indeed, that mon-oval twins are far from being invariably equipped with identical constitutions. Even apart from the effects of environmental influences in later stages, differences may arise during the very act of embryonic division upon which the production of mon-oval twins depends. This division, it would seem, does not always correspond to the first cell division of the fertilized ovum. If the critical division does not occur until a period of embryonic development when the differentiation of the organ rudiments has already begun, two very different fruits may arise, so that, for instance, one may be normal and the other an acardiac monster. Yet even in such extreme cases, the two fruits may have like hereditary equipments.

The question whether twins are mon-oval or di-oval cannot usually be decided by the condition of the foetal membranes at birth, for there seem to be exceptions to the rule that mon-oval twins have a common chorion, whereas di-oval twins have separate membranes. Besides, the condition of the membranes is not always recorded when twins are born. For practical purposes, however, a sufficiently certain diagnosis between mon-oval and di-oval twins can always be made by an examination of the pair in respect of certain characters whose heredity is known. Siemens and other observers tell us that the most important characters in this respect are: the tint of the hair, the eyes, and the skin; the distribution of the hair; freckles, telangiectases, keratosis pilaris; furrows in the tongue; the shape and the expression of the face; the shape of the ear; the shape of the hand; the build; the finger prints. Where we are dealing with coloured populations, the tint of the skin certainly does not help us very much. The shape of the head is to be accepted only with reserve as a criterion, for mon-oval twins often differ a good deal in respect of this character. To avert misunderstanding, it should be mentioned that the observer must not assume twins to be mon-oval because they are of the same sex. Of course, mon-oval twins are always of the same sex; but only about two fifths of all twins of the same sex are mon-oval twins.

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[1] Die Zwillingspathologie, Springer, Berlin, 1924.

In the case of monomeric characters, it is as a rule easy by the methods above described to reach a decision whether a particular character is partly or mainly determined by the hereditary equipment; but as regards polymeric characters, the decision may be very difficult. However, in the case of mon-oval twins we may generally expect to find that even polymeric characters (except such as are mainly determined by environmental influences) will be extraordinarily alike in both members of the pair; whereas such a close resemblance of polymeric characters in di-oval twins will be found only in a minority of the cases.

The study of twins, however, does not enable us to ascertain the degree and the kind of polymerism. On the other hand, the mere fact of the hereditary determination of characters frequently present, can in many instances be better elucidated by the study of twins than in any other way. For instance, it is mainly through the study of twins that we have learned how important hereditary predisposition is in the production of goitre (Siemens) and dental caries (Weitz). Only with very great caution must we infer from differences between mon-oval twins in respect of a character that this character is "non-hereditary". In my opinion, Siemens has gone much too far with his conclusions regarding birthmarks and left-handedness. We need, above all, to be cautious in respect of asymmetrical characters. There would seem, for instance, to be some reason to suppose that twin pregnancy is per se inclined to make one of the twins left-handed. H. H. Newman[1] has expressed the view that there is a resemblance in kind between the formation of the two halves of the body and the formation of twins; and C. H. Danforth[2] has drawn the inference that, in a sense, mon-oval twins may be regarded as differing from one another in the same sort of way as the right and left sides of an individual, so that it is not possible to refer the differences between them to environmental influences in the ordinary sense of the term. The conditions of embryonic development can, in like manner, lead unaided to asymmetrical growth; and the occasional "asymmetry" of mon-oval twins may be regarded as similarly determined.

We can use the correlation calculus for the numerical expression of the average degree of similarity in respect of a particular character as between mon-oval twins on the one hand and di-oval twins on the other. According to Siemens, a lack of correlation between mon-oval twins as regards a certain character, denotes that this character is not a hereditary one; whereas complete correlation does not denote that a character is completely hereditary (or very little determined by environmental influences) unless di-oval twins fail to display a more marked correlation in respect of this character than is displayed by sibs in general. It is, indeed, conceivable that the remarkable similarity of twins may be partly determined by the remarkable similarity of the environment in which the respective members of the pair develop. It would, however, in my opinion be too hasty an assumption were we to suppose that twins always develop under more closely similar environmental conditions than do other sibs. It is above all during the period of intra-uterine growth that mechanical environmental influences are more different in the case of twins than they are in the case of other sibs. The fairly extensive differences often observed as between the respective shapes of the skull in mon-oval twins may, for example, be due to the fact that twins necessarily occupy different positions in the womb, whereas a solitary foetus always has the whole space to itself. We must therefore avoid inferring that the shape of the head is not a hereditary character because we sometimes find a marked vertical extension of the head (hypsicephaly) in one member only of a pair of mon-oval twins.

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[1] The Biology of Twins, second edition, Chicago, 1924.

[2] Resemblance and Difference in Twins, "Journal of Heredity", vol. X, 1919, no. 9.

The mere fact that twins are of exactly the same age may partly account for the way in which they resemble one another more closely than do other sibs. Inasmuch as most of those who have made a special study of twins have been mainly concerned with observations on children, we must be careful to bear this source of fallacy in mind. During childhood, for instance, there usually occurs a darkening of the hair and an enlargement of birthmarks, and this may suffice to account for the fact that in twins such characters are more closely correlated than they are in other sibs.

If a character were exclusively dependent upon the hereditary equipment, we should expect to find in mon-oval twins a correlation of 1. If, on the other hand, environmental influences either favour or hinder the development of a character, the correlation of course falls below that amount. I have worked out a calculation to show the amount of the correlation to be expected in the case of mon-oval and di-oval twins respectively when a dominant heredity factor having a frequency of 0·5 has its manifestation hindered by environmental influences. If the hindrance operates

	in mon-oval twins	in di-oval twins
in 0·0 of the cases, the correlation is	1·00	0·42
,, 0·1 ,, ,,	0·69	0·29
,, 0·2 ,, ,,	0·50	0·21
,, 0·3 ,, ,,	0·37	0·15
,, 0·4 ,, ,,	0·27	0·11
,, 0·5 ,, ,,	0·20	0·08
,, 0·6 ,, ,,	0·14	0·06
,, 0·7 ,, ,,	0·10	0·04
,, 0·8 ,, ,,	0·06	0·02
,, 0·9 ,, ,,	0·03	0·01
,, 1·0 ,, ,,	0·00	0·00

The extent to which correlation is determined by the exceptional likeness of the environmental influences to which twins are respectively exposed may be ascertained by a comparison with the correlation between sibs examined at the same age as the twins under investigation. In conjunction with Meirowsky, examining 300 pairs of twins, I found that the correlation between mon-oval twins as regards the number of pigmentary naevi (the commonest birthmarks) to be 0·78 ± 0·036, and that of di-oval twins to be 0·31 ± 0·09. In this way correlation due to likeness of age is excluded. As the foregoing table shows, these correlations might be expected if the birthmark in question were to the extent of about $\frac{9}{10}$ths dependent upon the hereditary equipment. Fig. 164 shows curves representing the correlations to be expected. The curve for mon-oval twins applies also to intermediate and to polymeric behaviour. In the case of di-oval twins, however, there are differences here. If, instead of two variability classes, there were three or more, we should have to expect somewhat higher correlations (see pp. 547 and 586).

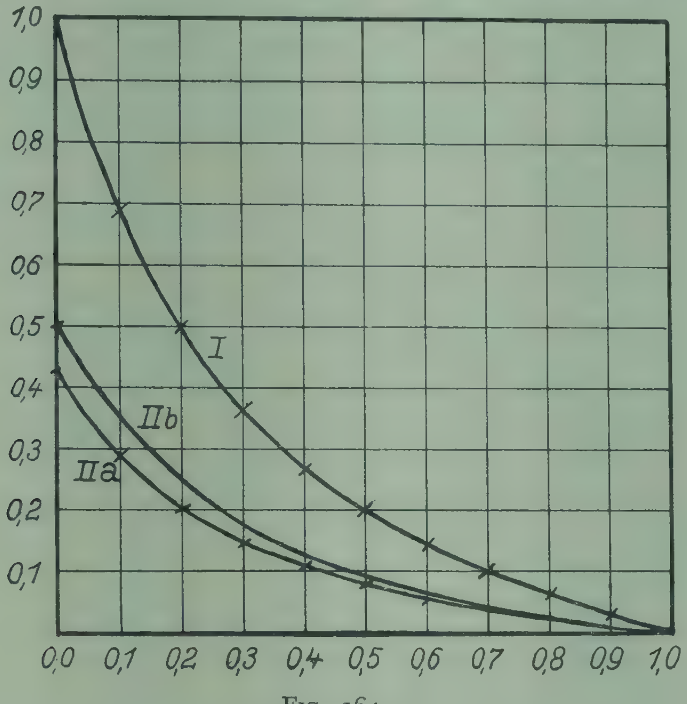

FIG. 164.

Correlations when the manifestation of a heredity factor having a frequency of 0·5 is to an increasing degree hindered by environmental influences.

Curve I. Correlation in mon-oval twins for a dominant or an intermediate factor.
Curve IIa. Correlation in di-oval twins for a dominant factor.
Curve IIb. Correlation in di-oval twins for an intermediate factor.

O. Verschuer[1] has used the mean percentage of deviation to illustrate the differences between twins or the resemblances between them. We ascertain the percentage deviation between the members of a pair of twins in respect of some particular character by recording the deviations of each of the twins from the mean of both twins in percentages of that mean. The mean percentage deviation of a number of pairs of twins is then obtained by calculating the arithmetical mean of the individual percentage deviations. The environmentally determined proportion of the total difference between di-oval twins can then be ascertained by dividing the difference between mon-oval twins by the difference between di-oval twins.[2] The difference between di-oval twins is partly determined by the hereditary equipment and partly by environmental influences, whereas the difference between mon-oval twins is determined only by environmental influences, and we may therefore assume that the environmentally determined difference between di-oval twins is just as great as the environmentally determined difference between mon-oval twins. Obviously, then, the before-mentioned fraction must indicate the proportional part played by environmental influences in producing the differences between di-oval twins. If we wish to estimate the share of environmental influences in producing the differences between other sibs, we must allow for the fact that the resemblance between twins as compared with the resemblance between other sibs is somewhat increased owing to environmental influences. We can try to ascertain this amount by comparing the differences between di-oval twins with the differences between other sibs, and then establishing a ratio between the differences between mon-oval twins on the one hand and the differences between ordinary sibs on the other (allowing for differences in ages). Finally, following up the method farther still, we can proceed to estimate the part played by environmental influences in the variability of non-related individuals in a population, by establishing a ratio between the average differences among mon-oval twins and the average differences among other individuals of the same ages.

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[1] Die Wirkung der Umwelt auf die anthropologischen Merkmale nach Untersuchungen an eineiigen Zwillingen, "Archiv für Rassenbiologie", vol. XVII, 1925, no. 2.

[2] O. Verschuer, Die vererbungsbiologische Zwillingsforschung, "Ergebnisse der inneren Medizin und Kinderheilkunde", vol. XXXI, 1927, p. 35.

The application of the mean percentage deviation is for the most part restricted to the study of continuously variable characters susceptible of measurement (for instance, stature, weight, etc.). It is not satisfactorily applicable even to such characters as the tint of the hair or that of the eyes, and it is of very little use for the study of the heredity of diseases. The twin method of study often encounters practically insuperable difficulties when we are studying the heredity of the rarer morbid states. For, as a rule, we are unable to find a sufficiency of twins exhibiting the character in question. The ratio of twin births to ordinary births is about 1 : 80, and that of mon-oval twin births to ordinary births only about 1 : 300 or 1 : 400. We can only apply the method to a character when we have a sufficiency of twin material.

That is why the twin method of study needs a comprehensive organisation, transcending the limits imposed upon the individual investigator and the individual institution. Siemens rightly demands that arrangements should be made for the expert study of all twins throughout the population, and for the establishment of "twin archives" in which such data can be permanently registered. The twins should be kept under lasting observation, and at intervals should be subjected to careful re-examination. A further desideratum is that the State should undertake a general and continuous *biological registration of the entire population.* In default of this, many of the data essential to the science of genetics can never be obtained in sufficient numbers or in a sufficiently trustworthy form. Not until such a registration has been undertaken shall we be able to achieve a trustworthy conception of the hereditary constitution of the population in general and of each family in particular. We must bear in mind, too, that such a registration would be of inestimable value to the sciences of hygiene and social hygiene as well as to the science of genetics. The cost would soon be repaid many times over by the resulting improvement in the health of the community. Furthermore, with the aid of the data thus acquired, far-reaching racial hygienic measures could be inaugurated.

SECTION FIVE

THE INHERITANCE OF INTELLECTUAL GIFTS

by

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CHAPTER TWELVE

THE INHERITANCE OF PARTICULAR TALENTS

THE mental differences between human beings are not only much more extensive than the bodily differences, but are also enormously more important. The civilised world, the world in which we have to live, is a product of the human mind. The mental equipment of various human beings, their capacity to preserve and to extend the acquirements of civilisation, varies greatly. Between the genius and the idiot there are countless intermediate grades of intelligence, and like considerations apply to differences of temperament and character. Why is it that many persons are able, many others stupid, and the majority mediocre? Why are some people cheerful and others gloomy; some industrious and others slothful; some unselfish and others selfish? Those who are accustomed to thinking biologically, have no difficulty in understanding that the mental characteristics no less than the bodily characteristics of human beings are rooted in the hereditary equipment, and that environmental influences (including education in the narrower sense of the term) can do no more than help or hinder the flowering of hereditary potentialities. But it is certain from the outset that differences in mental endowment exist, and the only question is to what extent environmental (non-hereditary) influences are able to modify the working of these hereditary factors.

In the psychological domain, therefore, on account of its decisive importance for civilisation, lies the core of genetics. Unfortunately, considered as a subdivision of genetics, this psychological field is the most difficult one to cultivate, for the reason that mental qualities and trends are so little accessible to measurement, and are, in other respects as well, hard to grasp for precise examination. Nevertheless, the methodology of psychological genetics differs in no respect from the methodology of genetics in general (see above, p. 504). "The basic fact of experience in the practical investigation of heredity is the psychical similarity between blood relations. The ascertainment and analysis of this, the discovery of its phenomenal laws, must always remain the foundation of a psychological science of heredity."[1]

In our account of morbific heredity factors we have already noted the existence of certain facts which justify conclusions regarding the heredity of normal mental capacities. Thus, from the sex-linked recessive heredity of red-green blindness, we were able to infer that certain sex-linked dominant heredity factors are essential to the production of normal colour vision. This inference is nothing more than a contemplation of the same facts from the other side. But colour blindness is a mental anomaly, namely, a defect in the power of sensual perception, whereas the capacity to distinguish all the ordinary colours is a normal mental faculty. In like manner, from the existence of recessive heredity factors which determine deaf-mutism we infer that there are certain dominant heredity factors which contribute to the production of normal hearing. It would be possible in a population where the great majority of persons were deaf-mutes to trace a normal capacity (or, rather, what is among ourselves the "normal" capacity) for hearing as a dominant hereditary trend; and, indeed, in such circumstances the power to hear as we call it "normally" would present itself as a peculiar and exceptional higher faculty enabling certain individuals to become aware of processes without seeing them—a faculty of which the great bulk of the population could form no true conception. From the existence of hereditary mental weakness, we can confidently infer that the normal powers of the understanding are hereditarily determined; and, further, from the fact that there are numerous different kinds of hereditary mental weakness, we can infer that a considerable number of genes must cooperate in the production of a normal understanding, and that the intelligence will be impaired if any one of these genes be lacking. Once more, in a population consisting predominantly of feeble-minded persons, the intelligence which we now call "normal" would stand out as the expression of exceptional endowments, whose nature would be utterly beyond the grasp of the great majority of the population.

[1] W. Peters, Die Vererbung geistiger Eigenschaften, Fischer, Jena, 1925, p. 30.

Similar considerations apply to the heredity of all "gifts" which transcend the mental endowments of our extant population to the same extent as that to which the mental endowments of ordinary people to-day transcend those of the feeble-minded. Such exceptional gifts are usually spoken of as manifestations of "talent" or "genius", which are rather vague terms. Generally by "talent" we understand a comparatively specialised high mental endowment, above all in the artistic field, whereas by "genius" we mean a many-sided and comprehensive endowment. But a difference of degree also comes into the question. Just as "idiocy" signifies a higher degree of feeble-mindedness than "imbecility", so "genius" denotes a more outstanding degree of mental endowment than does "talent". We consider, therefore, that among a million persons selected at random there will be a considerable number of "talented" individuals, but very few "geniuses".

I do not think it satisfactory to restrict the application of the term "genius" to persons whose gifts are peculiarly "creative". No doubt a person of genius is more likely than an ordinary human being to win new knowledge or to produce original works of art, but we should certainly be going too far if we were to describe as a "genius" everyone who does or produces something really new. In many cases such innovators, though gifted, are extremely one-sided persons. There are neither arguments nor facts to justify the notion that "genius" is a creative activity which manifests itself causelessly, independent of hereditary and environmental influences.

It will obviously be difficult to trace the heredity of ordinary, everyday differences in mental endowment; but as soon as we come to exceptional gifts—those which deviate more markedly from the average—our task becomes easier. Especially striking is the way in which *musical talent* tends to run in families, and for this reason the heredity of exceptional musical faculty has been very carefully studied.

In the Bach family, exceptional musical endowment can be traced in an unbroken line through five generations of males. Among the sons of Johann Sebastian Bach there were no less than five noted musicians; and among the other descendants of Johann's great-grandfather there were several other talented musicians. In the Bach family there was also a pair of twins (obviously mon-oval), namely, Johann Sebastian Bach's father Johann Ambrosius and the latter's brother Johann Christoph. Of these twin brothers, Philipp Emanuel Bach (a son of Johann Sebastian) writes in his memoirs: "They resembled one another so closely that even their wives could not tell them apart. . . . In speech, in mood, in everything, they were alike. In music, too, you could not distinguish them, so like was their execution, so like their interpretation. If one of them was ill, the other was ill also." Their deaths, likewise, occurred in brief succession.[1]

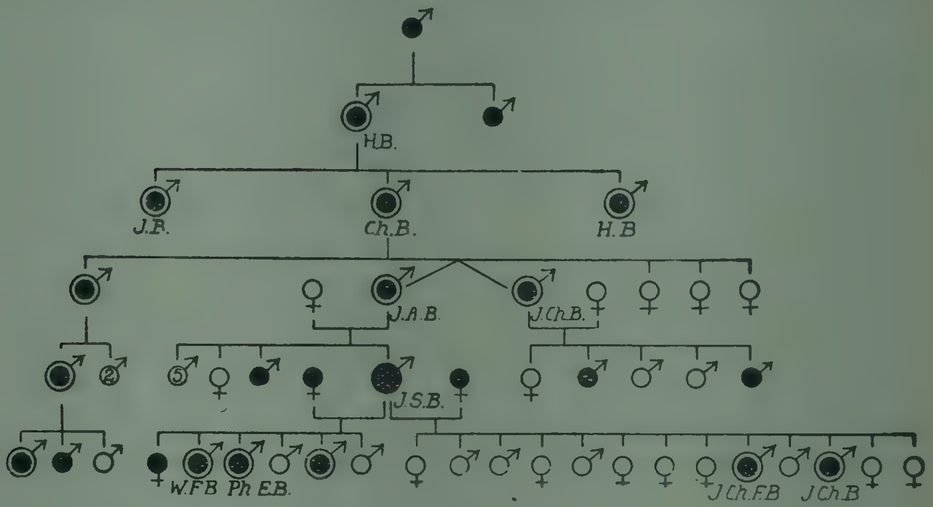

FIG. 165.

Exceptional musical talent in the Bach family. The black circles surrounded by rings denote celebrated musicians. The large black circle in the middle of the figure indicates Johann Sebastian Bach. The persons indicated by small white circles are not to be regarded as having been all unmusical, but it is not recorded that any of them had exceptional musical gifts. Among the 19 children of Johann Sebastian Bach, not one of them was altogether unmusical.

The genealogical tree shown in Fig. 165 is mainly derived from O. Feis.[2] I am also indebted for valuable genealogical details to the publisher of the present book, Dr. Friedrich Lehmann of Munich. As the table shows, Johann Sebastian was twice married, and in each case to a woman with musical gifts. In this matter I follow the account given by Jon Alfred Mjöen.[3] The first wife, Maria

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[1] Cf. Kekulé von Stradonitz, "Deutsche Herold", 1920.

[2] Studien über die Genealogie und Psychologie der Musiker, Bergmann, Wiesbaden, 1910.

[3] Genius as a Biological Problem, "Eugenics Review", vol. XVII, 1926, no. 4.

Barbara, was her husband's second cousin; husband and wife had a common great-grandfather in the male line. If anything above average musical endowment were to be indicated in the genealogical tree showed in Fig. 165, practically all the small white circles ought to have been black. The tree, therefore, shows not the distinction between the presence or the absence of ordinary musical gifts, but the distinction between very high musical endowments and endowments which are not so high but are still above average. Besides, it is likely enough that several instances of exceptional musical endowment may have remained unnoticed, especially in female members of the family.

In the Mozart family and in the Weber family (the two were interconnected by marriage) exceptional musical talent can likewise be traced through several generations. In Fig. 166, in the last generation, Wolfgang Amadeus Mozart is indicated by a black circle with a ring round it; in the penultimate generation, we see on the right hand, similarly denoted, his father Wolfgang Mozart, and on the left hand, likewise similarly denoted, Karl Maria von Weber.

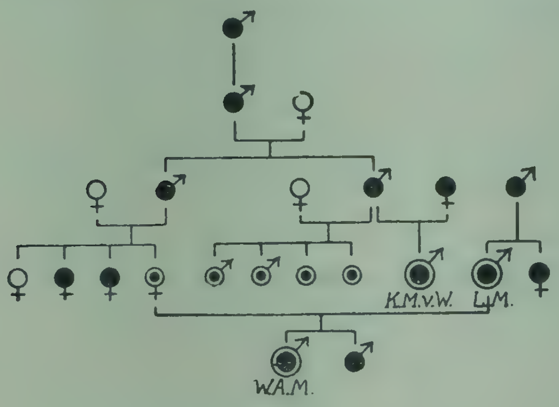

Fig. 166.

Musical talent in the Mozart-Weber family (after Mjöen). The musical individuals endowed with exceptional talent are denoted by black circles, and in the case of musicians of genius the black circle is surrounded by a ring; a point within a circle indicates average musical endowment; the unmusical members of the family are denoted by white circles.

H. Kurella[1] has collected the records of 28 musical families in which exceptional musical endowment can be traced through three or more generations. This applies to the

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[1] Die Intellektuellen und die Gesellschaft, Bergmann, Wiesbaden, 1913, p. 119.

Beethoven, Brahms, Schubert, and Liszt families, among others. Unfortunately Beethoven, Brahms, Gluck, Handel, Haydn, and Schubert remained childless. Had they had children, we may presume that the exceptional musical endowment would have been traceable in subsequent generations.

V. Haecker and T. Ziehen,[1] having circulated a questionnaire, received information relating to 350 families and found that the heredity factors producing musical talent seem, in general, to be dominant; but that there may, in some cases, also be supplementary recessive factors at work. Females are less often than males said to be highly endowed with musical faculty; nevertheless, in the case of equally gifted musicians, inheritance through the mother could be traced more plainly than inheritance through the father, from which it would appear that the heredity factors leading to the production of exceptional musical talent less often secure expression in females than in males. H. Koch and Fridtjof Mjöen,[2] however, having circulated a questionnaire, obtained information relating to 315 families which led them to the conclusion that positive and negative musical hereditary equipment in the parents are equally effective, and also that hereditary equipment in the father is no less effective than hereditary equipment in the mother. If this be so, the heredity factors producing exceptional musical talent must be neither dominant nor sex-linked.

J. A. Mjöen,[3] having secured information relating to 114 families, finds that when both parents are unmusical, their children are always unmusical, but that when both parents are highly gifted musically, they always have children with considerable or altogether exceptional musical endowments. Moreover, the intensity of the musical endowment in the children increases proportionally with the intensity of the musical endowment in the parents. In a Norwegian kinship, all the 37 members had exceptional musical gifts.

Very instructive is Mjöen's report concerning the kinship of the Norwegian composer Cleve. The first wife of this composer's father was an unmusical woman, who gave birth to five unmusical children. The second wife was a woman with musical talent, who gave birth to five musical children, among which was the musician of outstanding talent Halfdan Cleve. In one of the families on which Mjöen reports, a woman performer endowed with outstanding musical talent married to a musical man had seven extremely musical children, but she herself was said to be the child of two unmusical parents. Subsequently, however, Mjöen learned from a member of the family that she had been an illegitimate child, and that her father had really been a great musician.

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[1] Zur Vererbung und Entwicklung der musikalischen Begabung, Barth, Leipzig, 1923.

[2] Die Erblichkeit der Musikalität, "Zeitschrift für Psychologie", vol. XCIX, 1916, p. 16.

[3] Zur Erbanalyse der musikalischen Begabung, "Hereditas", vol. VII, 1925.

In this study of musical heredity, Mjöen did his utmost to check the records as regards the degree and the kind of musical endowment. The chief stress was laid upon the capacity for catching up a tune, upon that for reproducing one of the lower parts of a harmonised melody, and upon that for improvising such a lower part. In addition, Mjöen has endeavoured (as have also Haecker, Ziehen, and Stanton)[1] to analyse the various components of musical endowment.

Obviously, then, musical talent is not a unified faculty, but is an expression of the working of several different genes. Beyond question its causation is polymeric. Fridtjof Mjöen[2] has expressed the view that the basic element in it is sensibility to differences of pitch; but it is manifest that equally important are a keen receptivity for harmonies and discords, an affective sensibility to the sequence of tone (melody), and a sense of rhythm. But even if these various factors of musical talent are all combined in one individual, it does not follow that he or she will be a great musician. Marked musical sensibility is by no means rare in the feeble-minded, but it is self-evident that a feeble-minded person can never be a great musician. More especially we must remember that for creative activity in the field of music there is requisite, not only a good ear for music, but also a rich emotional life, a vigorous imagination, and a considerable power of intellectual production. That is why, no doubt, in the family of a musical "genius", we so often find a number of talented musicians, but hardly ever a second musical genius. We need not assume, therefore, that Johann

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[1] H. M. Stanton, The Inheritance of Specific Musical Capacities, "Eugenics Record Office Bulletin", 1922, no. 12.

[2] Die Bedeutung der Tonhöhenunterschiedsempfindlichkeit für die Musikalität und ihr Verhalten bei der Vererbung, "Hereditas", vol. VII, 1925.

Sebastian Bach very greatly excelled his musical relatives in respect of his musical endowments strictly considered. It was probably enough that there should have been superadded to his musical talents some further exceptional mental endowment. In other fields, likewise, the difference between the one-sided equipment of talent and the comprehensive equipment of genius doubtless depends upon the same sort of thing.

The frequency with which musical talent tends to run in families is favoured by the circumstance that musical people are apt to mate with persons of like tastes and endowments ("assortative mating," see above, p. 551). Galton drew attention to this matter, and all subsequent investigators have confirmed his observations.

Exceptional *talent for painting and sculpture* likewise tends to run in families. Among the kindred of Titian there were nine painters.[1] In the case of special endowment for the plastic arts even more than in the case of special endowment for music, peculiar intellectual gifts must be superadded to the more specific artistic gifts (a sense of form in the case of the plastic arts, corresponding to the "good ear" of the musician) if notable achievements are to be the result. Great painters and sculptors usually spring from families in which there have also been persons pre-eminent in other fields. This applies, for instance, to the family of Anselm Feuerbach.

As regards a talent for painting, marked visualising power (the so called "eidetic" gift) would seem to be of special importance. The psychiatrist R. Sommer[2] writes: "A plastic vividness of the visual imagination, such as in combination with other exceptional endowments often leads to notable achievements in the field of painting or sculpture or architecture, is apt to run in families, producing in this individual or in that a different effect according to the nature of the different intellectual tasks to which the individual addresses himself."

Very striking too are the manifestations of the heredity of *mathematical talent*.

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[1] F. Galton, Hereditary Genius, Macmillan, London, 1869.

[2] Familienforschung und Vererbungslehre, second edition, Leipzig, 1922.

Famous is the story of the mathematical family of the Bernoullis, to which at least 8 (or perhaps 9) notable mathematicians belonged. It is characteristic that, of these, several should, to begin with, have taken up some other occupation. One started upon a mercantile career; two studied law; and a fourth Bernoulli intended to become a biologist—but, nevertheless, three of these four became professors of mathematics, and the fourth a professor of physics.[1]

There would seem to be some correlation between mathematical and musical talent, for these two gifts are not infrequently found in the same individual or in different individuals belonging to the same family (Möbius, Kurella). Inasmuch as tone ratios depend upon numerical ratios, in musical perception there must to some extent be a process of unconscious counting at work. But the association of musical talent and mathematical talent is by no means invariable.

Davenport[2] has reported on a family in which various members displayed a remarkable talent for calculation.

In the Krupp family,[3] a talent for technical invention conjoined with a marked talent for business organisation can be traced through three generations in the male line. Going farther back than this, however, we find that the grandmother of Friedrich Krupp (the founder of the works at Essen) was an unusually gifted and energetic woman. Friedrich Krupp's wife was also a person of exceptional vigour and strength of will. In her son Alfred, who was only fourteen years of age when his father died, this energy of the mother was so happily combined with the inventive imagination of the father that, notwithstanding his extreme youth, he was able to carry on the business and to develop it enormously. In his son Friedrich Alfred, we likewise find a happy combination of inventive talent and organisational talent. In the Siemens family, among 14 sibs there were 3 (some say 4) brothers who were distinguished inventors and successful entrepreneurs. The most notable of these was Werner von Siemens, to whom we owe a considerable proportion of the great inventions of the nineteenth century. It is noteworthy that inventive talent existed, not only in the Ohlhöf branch of the family, but also in the Wedding branch, where it was manifested by three of the Siemens whose only tie of blood with Werner von Siemens was that they had a common great-great-grandfather. Moreover, the Siemens family has produced a considerable number of men who were enterprising and talented in various fields.[1]

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[1] P. J. Möbius, Ueber die Anlage zur Mathematik, Leipzig, 1907.

[2] Quoted by W. Scheidt, Allgemeine Rassenkunde, Munich, 1925.

[3] Krupp, 1812–1912, Fischer, Jena, 1912.

As long ago as 1869, in *Hereditary Genius*, Galton published notes on a considerable number of distinguished families, and his studies of this matter have since been supplemented by other investigators. Families celebrated in history and to which several noted military commanders or statesmen belonged were those of Alexander the Great, Hannibal, Scipio, Charlemagne, and William of Orange. In the family of the celebrated Dutch jurist Grotius, there were for four successive generations persons noted for their achievements in the intellectual field.

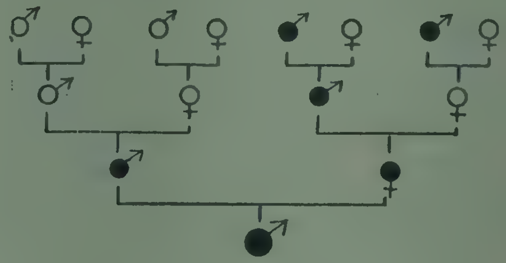

FIG. 167.

Above-average talent in Goethe's ancestral table.

Boyle, the celebrated chemist, physicist, and philosopher, had 14 near relatives with exceptional gifts; and James Gregory (1638–1675), the mathematician and the inventor of the reflecting telescope, had 15.[2] In England, from the interconnected families of the Pitts, the Temples, the Grenvilles, and the Wyndhams, there issued a remarkable number of leading statesmen. Talented almost without exception were the members of the Sidney, Montagu, and North families, which for ten successive generations continued to intermarry.

In a second book, Galton[3] reported concerning a large number of English scientists and their descent. A third volume[4] contains the family histories of 100 Fellows of the Royal Society. Especially remarkable were the data concerning the Balfour family, which has produced a large number of leading statesmen, investigators, men of learning, entrepreneurs, and successful merchants.

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[1] Cf. H. W. Siemens, Ueber das Erfindergeschlecht Siemens, "Archiv für Rassen- und Gesellschaftsbiologie", 1916–1917, no. 2.

[2] Cf. Agnes Grainger Stewart, The Academic Gregories, Famous Scots Series, Oliphant, Edinburgh, 1901.

[3] English Men of Science, their Nature and Nurture, London, 1874.

[4] F. Galton and E. Schuster, Noteworthy Families, London, 1906.

Goethe is usually spoken of as an instance of the isolated appearance of great gifts. The family history of Goethe is fairly well known.[1] It would seem that Goethe's unrivalled artistic imagination, his capacity for insight into the minds of others, and his high intelligence, were mainly inherited from the maternal side. I shall, however, come back to the question of how far certain factors derived from the paternal side may have exercised an important influence upon Goethe's achievements.

For the rest, Goethe's talents were not so universal as is commonly assumed. It is true that he effectively realised that a knowledge of nature must be the foundation of mental culture, but his own gift for scientific research was not exceptional. He had the seer's vision, but the strict methodology of natural science was alien to his temperament. Nor had he any special aptitude for mathematics. The human intermaxillary bone, whose discovery is often ascribed to him, was known to earlier anatomists (such as Vesalius and Fallopius),[2] although before him no one had laid much stress upon it.

A very striking example of a highly gifted family is that of the Darwins and the Galtons. Francis Galton was Charles Darwin's first cousin, and Galton's intellectual gifts were hardly less remarkable than those of Darwin. The fact that Darwin's discoveries produced a sort of earthquake in the world of ideas and that Galton's discoveries did not, must not be taken as signifying that the latter was less gifted than the former; and in any case Galton was the founder of racial hygiene. In the domain of genetics, indeed, he had clearer notions than had his cousin Darwin, and he can be placed beside Mendel as a pioneer in the modern study of heredity. In addition to Charles Darwin and Francis Galton, there were a number of distinguished persons in the same kinship; and the two men's common grandfather Erasmus Darwin conceived the fundamental notion of evolution earlier than Lamarck.

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[1] R. Sommer, Goethes Wetzlarer Verwandtschaft, Barth, Leipzig, 1908; Goethe im Lichte der Vererbungslehre, Barth, Leipzig, 1908.

[2] L. Plate, Jenaer Professoren als Förderer der Abstammungslehre, "Verhandlungen der Deutschen Zoologischen Gesellschaft", 1925, p. 17.—When Spengler, in his Untergang des Abendlandes, declares that Goethe's discovery of the intermaxillary bone outweighs all Darwin's achievements, he is merely displaying his own ignorance.

"One of the most striking illustrations of the inheritance of ability is afforded by the descendants of Erasmus Darwin. On the originality, general ability, and productiveness of Erasmus Darwin it is not necessary to dilate. Robert Waring Darwin, his son, was a distinguished physician, and, like his father, an F.R.S.

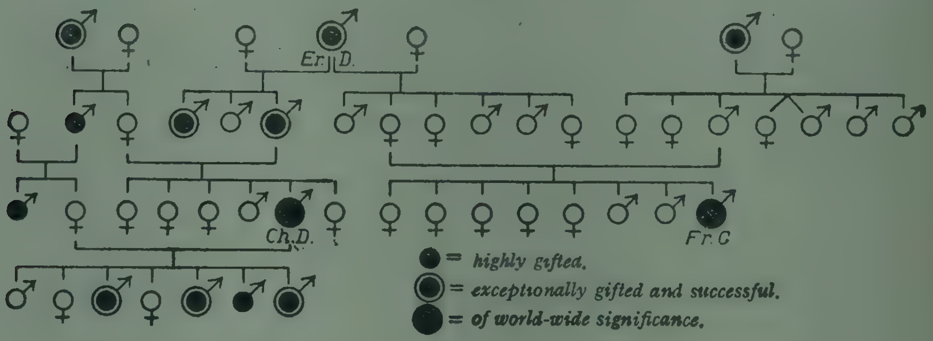
Er. D.
Ch. D.
Fr. G
● = highly gifted.
◉ = exceptionally gifted and successful.
⬤ = of world-wide significance.

FIG. 168.

The Darwin, Galton, and Wedgwood families. The persons signalised in the above family tree as having exceptionally high gifts are not to be regarded as constituting an exclusive selection. Presumably other members of the families of these were also highly gifted, especially among the women, who, in existing social circumstances, had less opportunity for manifesting their endowments.

Another son, Charles, was a man of remarkable promise, and, although he died at the age of twenty, he gained the first gold medal of the Aesculapian Society for experimental research. Charles Robert Darwin, the author of the *Origin of Species*, and by common consent one of the world's greatest men of science, was the son of Robert W. Darwin. He married his first cousin, Emma Wedgwood, a granddaughter of Josiah Wedgwood, F.R.S., the founder of the pottery works that produced the famous Wedgwood ware. Charles Darwin's four sons became men of note: Francis Darwin, F.R.S., a prominent English botanist; George Darwin, F.R.S., noted astronomer, and professor of astronomy at Cambridge; Horace Darwin, F.R.S., a prominent engineer; Major Leonard Darwin, author of works on political economy, president of the Eugenics Education Society, and president of the International Eugenics Congress."[1]

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[1] S. J. Holmes, The Trend of the Race, New York, 1921.

Obviously in such cases we are not simply concerned with one dominant heredity factor, which diffuses itself throughout a kinship as does the factor producing six fingers on a hand or the factor producing night-blindness. The heaping-up of persons of talent in such kinships must be largely induced by the fact that distinguished families are prone to intermarry.

A good instance of two interconnected families is that of the Ficks and that of the Wislicenus, which have produced a remarkable number of leading scientists and men of learning. A Fick has reported upon them.[1] No doubt there must be in Germany many similar families which are only waiting to be described from the outlook of hereditary talent. If in the case of many such families no remarkable achievement has been recorded in earlier generations, this must chiefly be because opportunities for the exercise of exceptional gifts were lacking. When we trace the ancestry of urban families, we find that in the great majority of instances they came only a few generations ago from the countryside—and in country life, especially in agricultural occupations, there is little opportunity for the display of exceptional talent.

Galton[2] made a statistical enquiry relating to the kinships of 415 celebrated men. In the 300 families to which these belong, there had been about 1000 men of note; while it appeared that, on the average, 100 celebrated men had had 31 noted fathers, 41 noted brothers, 48 noted sons, 17 noted grandfathers, and 14 noted grandsons. These figures are frequently quoted, but those who quote them usually fail to make it clear that Galton attached a very rigorous notion to this term "exceptional talent", considering that among a million persons there were only about 250 highly gifted men—this being a proportion of 1 : 4000. It follows that, if talent were not hereditary, among the 100 fathers of 100 celebrated men we should expect to find only 0·025 celebrated men. In this matter, then, experience outdoes expectation by 1240 : 1. Again, among the 200 grandfathers of 100 celebrated men, we should expect to find only 0·05 celebrated men, whereas Galton found 17. The reason why there is a considerably smaller proportion of celebrated persons among the grandfathers than among the fathers of celebrated persons is that a man has a considerably smaller proportion of his hereditary equipment in common with either of his grandfathers than with his father. Since, on the average, 100 Fellows of the Royal Society have 206 brothers, we should expect, if the distribution of talent were a matter of pure chance, to find among the brothers of 100 celebrated men about 0·05 celebrated brothers, whereas actual experience showed that the number was 800 times as great as this. If we assume that 100 celebrated men of that day had 200 sons, we find that the actual distribution of celebrated persons among the sons of celebrated persons exceeds the chance expectation by nearly 1000 : 1.

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[1] Die Familie Fick, "Archiv für Rassenbiologie", vol. XIV, 1922, no. 2; Die Familie Wislicenus, ibid., vol. XV, 1923, no. 2.

[2] Hereditary Genius, p. 477.

In the United States, F. A. Woods[1] has traced the kindred of 3500 noted American citizens. Whereas any American citizen, taken at random, had a 1 in 500 likelihood of being closely related to one of these, the actual probability that one of the celebrated men would be closely related to another of the celebrated men was 1 in 5—that is to say a hundredfold the calculated probability. On the average, each of the 46 celebrated Americans who had acquired a special place of honour in the Hall of Fame had had more than one distinguished relative. These ratios ranging from 1 : 100 to 1 : 500 are considerably lower than those derived from Galton's data, which range from 1 : 800 to 1 : 1200. The probable explanation of this difference is that in the democratic United States there is not so marked a heaping-up of talented persons in particular families as in Britain where class distinctions are more scrupulously maintained, but that in America there is a more thorough and persistent shuffling of the classes and the families. In part, moreover, the greater heaping-up of persons of talent in certain English families as compared with American families may be a semblance rather than a reality, for it may depend upon there being a greater furtherance of the manifestation of talent thanks to family influence, etc., in Britain, whereas in America the individual is thrown more upon his own resources. The latter circumstance may not be an unconditional advantage, for we may very well conceive that the result may often be that a highly talented person will fail to secure the appropriate environment for the development and manifestation of his gifts.

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[1] Heredity and the Hall of Fame, "Popular Science Monthly", vol. LXXXII, 1913.

D. R. Brimhall,[1] studying data relating to 956 noted American men of science (including mathematicians and psychologists), obtained similar results to Galton and Woods. It transpired that a very large proportion of the distinguished relatives of distinguished persons had attained distinction in the same specialty as that pursued by the individual who had been the starting-point of the investigation. Thus, among 82 distinguished relatives of distinguished physicists, 18 had also attained distinction in the domain of physics; and among 36 distinguished relatives of distinguished botanists, 12 were themselves distinguished botanists. Noteworthy, too, is Brimhall's observation that the wives of noted men of science actually have more distinguished relatives than their husbands have. Obviously, then, marriage between the members of talented families must be comparatively common. Among 21 women who were noted for scientific achievements, 10 had married zoologists, which may signify that zoologists have a specially keen eye for the value of hereditary endowment. From his investigations, Brimhall draws the practical conclusion that it would be of great advantage to society at large were it to provide distinguished men of science with facilities for the procreation and upbringing of a sufficient number of children, inasmuch as of these children a much larger than average proportion might be expected to do work of exceptional advantage to a civilised community.

The inheritance of intellectual gifts is often denied on the ground that persons of genius very rarely have sons similarly distinguished. The fact is incontestable, but does not in any way militate against the belief that mental endowments are hereditarily determined. We have already learned that a considerable number of heredity factors must collaborate in order to produce an outstanding and comprehensive mental equipment. Now, every child receives from each of its parents no more than one half of that parent's hereditary equipment—

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[1] Family Resemblances among American Men of Science, "American Naturalist", vol. LVI, 1922, and vol. LVII, 1923.

and not by preference the better half of the equipment. Consequently, even if as a rule the wife of a man of genius were to have as outstanding a hereditary equipment as her husband (and in practice, of course, this is extremely rare), we should still have no reason to expect that the sons of men of genius would be geniuses like their fathers. Like all polymeric qualities, genius is not in mixed populations inheritable as such, but nevertheless the individual heredity factors which collaborate in its production are themselves hereditarily determined. If we were to adopt appropriate selective measures and to breed pure lines, genius would be hereditary like other polymeric qualities, and we could therefore "breed" geniuses.

One important reason why men of genius so rarely have sons who are also men of genius is that so many men of genius have very few children or none at all. On p. 570, I mentioned a number of noted musical composers who had no children. To this list of childless men of genius may be added the names of Michelangelo, Raphael, Kant, Frederick the Great, Hölderlin, Schopenhauer, Alexander von Humboldt, Grillparzer, Anselm Feuerbach, Klinger, and Nietzsche. Havelock Ellis[1] has devoted special attention to the tendency of men of genius to remain unmarried or to have few or no children. This is partly due to the peculiarities of their type, but partly also to social conditions. Kurella's view that the essential cause of the phenomenon is the peculiar disposition of persons of genius, is certainly one-sided. But whatever the explanation, the fact remains that the hereditary equipment which produces genius is apt to be destroyed almost immediately after it has been produced—and surely this could be avoided.

Persons who are prone to let words run away with them are fond of saying that genius is a mutation, using the term "mutation" to imply that a mysterious creative energy must be at work. Now, apart from the fact that mutations have not per se anything creative about them (see above, p. 480), such a view is, to say the least of it, distorted. As a rule, a mutation affects only one heredity factor, but genius unquestionably depends upon the collaboration of numerous heredity factors. It would be truer then, to say of genius, that it is a

[1] A Study of British Genius, London, 1904.

combination whose various elements are heredity factors which must at one time or another have come into existence by way of mutation.

Hereditarily determined exceptional talent sometimes (though by no means always) manifests itself at a remarkably early age. Mozart was already composing music at the age of six; Beethoven's first composition was printed when he was twelve; Mendelssohn wrote the overture to Shakespeare's "A Midsummer Night's Dream" when he was in his teens. Little children will sometimes show an extraordinary capacity for calculating (cf. the famous "calculating boy"). For great achievements in most other fields, however, experience is requisite—such experience as a child cannot have had. Still, Calderon wrote poems when he was fourteen, and Pope when he was sixteen. Schiller had written *The Robbers* by the time he was twenty-three; and at the age of thirty-four, Shakespeare had written most of his works. We owe to Miss Cox a comprehensive study of the manifestations of talent during the early youth of 300 distinguished men.[1]

In most of the departments of mental life, exceptional achievement must be the outcome of the joint working of a considerable number of favourable elements in the hereditary equipment: imagination, power of judgment, the capacity for distinguishing clearly between ideas, a gift for combination, a sense for truth, constancy of will, energy, ready apprehension, a good memory. The general "gift" will, of course, vary according to the nature of the various elements and the extent to which this element or that predominates in the compost. For a man of learning, receptivity is even more important than ready comprehension and a good memory. There are certain families in which many or all the members have remarkably good memories, and in these families there are many persons of distinction, especially men of letters, historians, and theologians. A usual accompaniment of good memory is a lively interest in details, that is to say in the historical aspect of things; and with this interest must to some extent be contrasted a special interest in generalities and in the formulation of scientific laws. The latter is what is essential to the investigator and discoverer, in addition to a flair for the essential and to a capacity for the intuitive grasp of causal relationships. A fertile imagination is indispensable, not only to the artist, but also to the creative thinker and the investigator. But in the scientist the imagination must be kept within bounds by vigorous powers of self-criticism, for in default of this our would-be scientist will be a mere visionary. Imagination provides the raw material of knowledge, just as mutation provides the raw material for the origination of species. But just as selection decides which among mutations shall become part of the permanent equipment of the species, so do the critical faculties decide which among the ideas that surge up out of the imagination can be real contributions to knowledge (see above, p. 498). We might, indeed, speak of a "theory of selection" in the domain of knowledge. The philosopher must have an incorruptible sense of truth, and must never shrink from unpalatable verities. The creative thinker must also be endowed with the power of freeing his mind from the prejudices of the day; and even the "spirit of contradiction" which all authorities with good reason dislike, may, in certain circumstances, be advantageous to the advance of knowledge. The entrepreneur, the statesman, and the military commander must be endowed with a good knowledge of human nature, with a vigorous will to power, and with exceptional foresight. The creative artist, the poet, and the man of letters must have a lively imagination and a well-developed plastic faculty, together with exceptional powers of putting themselves in others' places (empathy, or imaginative insight). The beautiful is what pleases. Hence the artist must have an instinctive feeling for what will please the public—and for that very reason, it will be hard for him to be at the same time a great thinker or a great investigator. Very favourable to exceptional achievement is good bodily health, without which strenuous and persistent mental work is scarcely possible. Absolutely indispensable is a lively interest in some particular field of knowledge or performance; and this interest will in most cases be made still more effective by a powerful impulse to self-assertion, generated by a strong mental or material will to power. The "ambition" or "vanity" of great men is almost invariably spoken of with disapproval, and yet there can be no doubt whatever that the trend denoted by these terms (for which it would be better to use the morally indifferent term "impulse to self-assertion"), is, though often unconsciously, one of the most important stimuli to great achievement. The objection will perhaps be made that such achievements are not "truly" great. Now, greatness in this sense is a concept of value of a kind which does not properly come within the domain of science. In the field we are now considering, the main thing we can register is the degree of celebrity. But worldly success and celebrity depend, not only upon actual achievement, but, and above all, upon an imposing appearance, an intuitive sense of what will impress the multitude, a facile pen or a ready tongue—and also upon cunning, the art of misrepresentation, and unscrupulousness.

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[1] C. M. Cox, Early Mental Traits of Three Hundred Geniuses, Terman's Genetic Studies of Genius, vol. II, Stanford University Press, 1926.

But whereas in the domain of certain "mental and moral sciences" we still find to-day as was the case in the time of Schelling and Hegel that celebrity is occasionally the outcome of a power to "talk big" and to posture effectively before the footlights, this danger can hardly be said to exist in the domain of the natural sciences. For this reason, it would be of great interest to undertake a genealogical study of such men as Gauss, Helmholtz, Clerk Maxwell, Heinrich Hertz, Boltzmann, Lorentz, Emil Fischer, Planck, Einstein, Bohr, T. H. Morgan, etc. W. Ostwald[1] has made a move in this direction.

The phenomenon known as "greatness" can be very variously determined. Many of the great figures of history are by no means distinguished for exceptional powers of judgment, and in many cases they are not even distinguished for exceptional understanding. Nor is character a necessary factor of greatness. We all know plenty of persons of blameless character whom no one would ever think of as great; and, on the other hand, not a few great men have had peculiarities of character which in ordinary mortals would be regarded as serious defects. Historical greatness is certainly not a unitary character. We shall have subsequently to refer to "greatness" as a phenomenon of mimicry. The claim to describe an individual as "great" will least often be challenged when it is based upon exceptional achievement in the field of the exact sciences or upon the manifestation of some such markedly specific talent as the creative musical faculty. As a rule, indeed, we do not call such specialists "great" unless their mental capacities in other respects are also well developed. Still, we cannot generally expect to find that a person who is exceptionally endowed in one direction will also be exceptionally endowed in many others. Those who have an extraordinarily dependable memory are often markedly devoid of critical faculty in matters which do not form parts of positive knowledge; and, on the other hand, persons with excellent powers of judgment may have their efficiency much impaired by weakness of memory. We can, indeed, from the existence of any exceptional talent infer with considerable probability that the mental hereditary equipment will be favourable in other respects, but there is not a close correlation here. Nay, we sometimes find as regards individuals a phenomenon which must be placed side by side with what is known as "reversion".

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[1] Grosse Männer, Studien zur Biologie des Genies, Akademische Verlagsgesellschaft, Leipzig, 1910.

CHAPTER THIRTEEN

EVERYDAY DIFFERENCES IN TALENT

The inheritance of moderate gifts is, of course, much less striking than the inheritance of exceptional gifts. Still, the inheritance of moderate gifts is not only a matter of daily experience, but can be proved by scientific methods.

The psychologist W. Peters[1] has examined the school reports of a great number of children, and has compared these with the school reports of their parents and grandparents. The reports relating to a particular child during successive school years and from various teachers were in general remarkably alike, so that inferences drawn from average values may be regarded as trustworthy. The reports of children on the average vary from the mean in the same direction as the reports of their parents; and it was found that on the average the deviation of a child's report was about one third as extensive as that of the child's parents. A comparison of the school reports of the grandparents with those of their grandchildren showed a conformity scarcely less extensive. Peters, taking the reports of children whose parents' reports showed similar averages, compared the children's reports with those of the grandparents, and found that the deviation of the children's reports from the mean was in the same direction as the deviation of the grandparents' reports. Seeing that, as far as grandparents and grandchildren are concerned, there can be little question of the effects of similar environments, we discern here a remarkably strong indication of the heredity of talent. Indeed, as the outcome of his investigations, Peters comes to the conclusion that environment (home education, etc.) has very little influence upon achievements at school. Peters has also applied the method of psychological measurement to the study of the gifts of a great number of sibs. The results of this investigation were similar. When the school achievements of the two parents had differed considerably, the children of these parents were found on the average to differ among themselves more markedly than when the achievements of the two parents had been approximately identical. Peters considers that this affords evidence of mendelian disjunctions.

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[1] Ueber Vererbung psychischer Fähigkeiten, Leipzig, 1915.

As early as 1903, Karl Pearson[1] endeavoured to appraise the mental resemblances of sibs through a study of 3000 reports on schoolchildren by their teachers. At first he applied his dubious four-fields coefficient as measuring rod, but he has recently discovered a correlation of $r = 0{\cdot}52$ in respect of the mental likenesses of sibs.[2] He had previously found the same correlation of $r = 0{\cdot}52$ as an average in the case of a number of the physical qualities of sibs. We are, therefore, entitled to say that the mental likenesses of sibs are as great as the bodily likenesses. A regression or "heredity figure" of $\frac{1}{3}$ cannot (as we have learned in the section on methodology) be regarded as implying that heredity is less influential (see above, pp. 550 and 551). When the choice of a partner in marriage has been made regardless of the question of talent, we may, if the behaviour of the heredity factors concerned be dominant, expect at most a regression of 0·33; and if the behaviour be intermediate, a regression of 0·5. Inasmuch as gifted persons, however, tend to intermarry, we should expect to find a higher regression when the talents are fully hereditary. But a regression of even as much as about $\frac{1}{3}$ signifies that the importance of heredity is here enormously greater than that of environment.

The following correlation table (see opposite page) is based upon the school reports collected by Peters relating to 3952 families.

From this table, W. Weinberg[3] deduces a correlation co-efficient $r = 0{\cdot}29$ and a correlation index $k = 0{\cdot}30$.

The alternative classification gives, on the foundation of the four-fields table, $r = 0{\cdot}23$ and $k = 0{\cdot}23$. We see that this alternative arrangement makes the correlation appear too small (see above, p. 538). On the other hand, Pearson's four-fields coefficient gives 0·46, this being a much higher value that the correlation coefficient derived from the detailed table, and being therefore presumably an improperly high value.

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[1] On the Inheritance of Mental and Moral Characters in Man, and its Comparison with the Inheritance of Physical Characters, "Journal of the Anthropological Institute", vol. XXXIII, 1903, p. 179.

[2] Karl Pearson, Inheritance of Psychical Characters, "Biometrika", vol. XII, 1919.

[3] Methoden und Technik der Statistik, in Gottstein's Handbuch der sozialen Hygiene, Springer, Berlin, 1925, vol. I, p. 432.

Weinberg has also drawn up a four-fields table which shows the correlation between the reports of the two parents. This gives a correlation coefficient of 0·26. It would seem, then, that in the choice of a partner in marriage, identical or similar degrees of talent coincide fairly often.

		Marks of the Children					No. of Families	Average of the Children
		1	2	3	4	5		
Marks of the Parents	1·0	177	198	51	—	—	426	1·70
	1·5	261	513	225	6	1	1006	1·98
	2·0	206	498	283	19	6	1012	2·13
	2·5	115	366	299	21	13	814	2·33
	3·0	54	177	191	8	13	443	2·43
	3·5	12	42	64	5	1	124	2·52
	4·0	9	29	48	4	2	92	2·58
	4·5	2	6	14	2	—	24	2·67
	5·0	—	3	6	—	2	11	3·09
No. of Families		836	1832	1181	65	38	3952	—
Average of the Parents		1·74	2·03	2·36	2·55	2·80	—	—

Pearson's collaborators, Schuster and Ethel M. Elderton, have likewise found the mental resemblances between sibs (schoolchildren) to be as great as the bodily resemblances.[1] They have also compared Oxford University reports on undergraduates in the case of fathers and sons, and have found a correlation in respect of mental characters which lags only a little behind the correlation of physical characters. In the case of the reports on brothers, the correlation was about $\frac{1}{3}$ higher than that between fathers and sons—which points to the co-operation of recessive heredity factors. In the United States,

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[1] E. Schuster and Ethel M. Elderton, The Inheritance of Ability, "Eugenics Laboratory Memoirs", I, London, 1907.

E. L. Thorndike[1] and his fellow workers found among sibs in respect of school achievements a correlation of $r = 0{\cdot}32$. All these correlations are approximately as high as could be expected to occur on the proviso of casual (unselective) mating if the hereditary equipment were alone determinative. We must, therefore, assume that the correlation partly depends upon a preference for a person having like endowments as a partner in marriage. We can certainly assume from the data of the correlation method that mental qualities are quite as markedly hereditary as bodily ones.

Especially interesting are the investigations made by Miss Gordon in the case of 216 pairs of sibs in Californian orphan asylums, for in the case of orphans living in public institutions the differences in mental capacity cannot be caused by differences in domestic environment. The children were examined in respect of their talents by the Binet-Simon tests as improved by Terman. As the outcome of the tests, each child was assigned a particular "intelligence age". The percentage ratio of this "intelligence age" to the actual age gives the "intelligence quotient". Now, as regards their intelligence quotient, the pairs of sibs exhibited a correlation of $r = 0{\cdot}61$. This must certainly be in part due to the fact that the sibs were, on the average, more closely alike in age than were the average children in the material. Miss Elderton[2] has tried to exclude the age element in this correlation and has worked out a corrected correlation of $0{\cdot}54 \pm 0{\cdot}24$. This value is likewise higher than would be expected in the case of unselective pairing, and it must therefore also be in part determined by a similarity of talent as between the parents.

In Germany, Max Schmitt[3] has studied the problem of the influence of environment upon the development of the intelligence. Examining orphans in institutions, he has come to the conclusion that no such influence is demonstrable. Reiter and Osthoff, making collective enquiries concerning children at schools for the feeble-minded (see above, pp. 428 et seq.), have found that the school achievements of sibs growing up in the same unfavourable environment were in about half the cases markedly different, which speaks in favour of the occurrence of mendelian disjunction and against attaching any considerable importance to environmental influences. The unfavourable environment to which these children are exposed is itself rather to be regarded as mainly the consequence of the defective mental endowments of the parents.

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[1] Heredity, Correlation, and Sex Differences in School Abilities, "Columbia University Contributions to Philosophy", 1903.

[2] E. M. Elderton, A Summary of the Present Position with regard to the Inheritance of Intelligence, "Biometrika", vol. XIV, 1923, nos. 3 and 4.

[3] Der Einfluss des Milieus und anderer Faktoren auf das Intelligenzalter, "Fortschritt der Psychologie", 1919.

G. Heymans and E. Wiersma[1] having, in response to a circular enquiry, received data relating to 437 families with 1541 children, were enabled to trace the heredity of 181 among 185 mental qualities regarding which enquiries had been framed.

During the examination of children living in a uniform environment, like that of an orphan asylum, it is especially those differences which are due to differences in hereditary equipment that come to light, whereas, environmentally determined psychological differences are seen in their purest form in persons having identical hereditary equipments, such as mon-oval twins. Francis Galton, who in these matters was so much in advance of his time, studied the life histories of a number of twins.[2] He recorded twenty instances in which twins of the same sex were notably different in their mental endowments. In none of these cases could exposure to like environmental conditions compensate the differences in mental constitution. The resemblances produced by subjecting both members of a pair to like educational influences were confined to externals, such as acquired knowledge and practice in certain accomplishments. It is true that in some instances in which sibs were kept almost entirely separated during childhood, considerable differences were manifest in adult life, yet it is noteworthy that these differences were not the outcome of the ordinary influences of everyday life, but of some such powerful cause as severe illness or accident. In the other cases, the resemblance persisted throughout life. Although many of Galton's informants were clergymen, a permanent change of character consequent upon a desire for self-improvement was not reported in a single one of these cases.

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[1] Beiträge zur speziellen Psychologie auf Grund einer Massenuntersuchung, "Zeitschrift für Psychologie", 1906 and 1907.

[2] The History of Twins, etc., see above, p. 554, footnote.

> A father of twins reported: "They have been brought up exactly alike since birth; they are both perfectly healthy and vigorous, but are in other matters as unlike in respect of bodily, mental, and emotional qualities as twin brothers could possibly be." Of another pair of twins we read: "They are not in the least like one another, either bodily or mentally, and the difference between them increases from day to day. They have always been exposed to the same environmental influences, and have never lived apart."

Many other cases have been reported in which di-oval twins differed widely from one another in respect of talent and character. Hitherto, however, not a single instance has been reported in which mon-oval twins (that is to say twins having the same hereditary equipment) have manifested extensive mental differences. The American racial hygienist P. Popenoe, has reported on an extremely interesting case in which twin sisters were brought up under markedly different environmental conditions.[1] These twins, whose mother died within a few days of their birth, were entrusted to different foster-parents when they were only a fortnight old. One of the sisters, Bess, was brought up by foster-parents belonging to the class of urban operatives; she attended school for only four years and then went to work as a commercial clerk and secretary; this occupation involved a great deal of travelling, sometimes in foreign lands. The other sister, Jessie, was brought up by foster-parents on a farm; she attended higher school and went to the university; after being a teacher for a short time, she married and had a child; then she resumed the teaching profession. Now, although these two young women had been exposed to such different environmental influences during the years of development, there were no notable differences in their character or their attainments; they were both equally well endowed and had the same trends and dispositions. Both of them were much liked by their associates; and both of them were vigorous and hard-working persons with indeed a tendency to overwork. Professor H. J. Muller,[1] of the University of Texas, had these twin sisters examined by the psychologist Miss Koch in regard to their mental endowments. Both of them shaped very well. In the alpha tests, such as are applied in the United States army, Bess got 156 marks and Jessie 153; in the Otis tests, which are applied for the measurement of higher intelligence, Bess got 64 marks and Jessie 62. We see, then, that in this respect a higher school and university education had not given Jessie any advantage over Bess. The probability that two persons should, as a mere matter of chance, be so much alike when subjected to intelligence tests is less than 1 : 2500. Furthermore, the results of the examination of these two cases go far to show that the intelligence tests really enable us to measure the hereditary equipment in this matter of talent without any notable vitiation by the amount of acquired culture.

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[1] Twins reared apart, "Journal of Heredity", vol. XIII, 1922, no. 3.

E. L. Thorndike[2] has examined 50 pairs of twins by psychological methods, and has discovered in them more than twice the amount of similarity that is usually found among sibs. Whereas, in the case of ordinary sibs, he found correlations ranging from 0·29 to 0·32, among twins he found correlations ranging from 0·69 to 0·90, the average being about 0·78. Unfortunately, he did not, in his investigation, distinguish between mon-oval and di-oval twins. If we assume one fourth of the twins to have been mon-oval, and if we assume mating to have been unselective and environment to have had no influence, the expected correlation would have been at most 0·60. The fact that Thorndike found higher values must, as Peters[3] has pointed out, be primarily referable to the sets of twins being of the same age (see above, pp. 557 et seq).

W. Weitz[4] reports concerning the mon-oval twins examined by him: "In almost all instances, the mon-oval twins were extraordinarily alike in respect of psyche; in respect, that is to say, of character, temperament, and mental endowments." Again,

[1] Mental Traits and Heredity, "Journal of Heredity", vol. XVI, 1925, no. 12.

[2] Op. cit.

[3] W. Peters, Die Vererbung geistiger Eigenschaften, Fischer, Jena, 1925, p. 283.

[4] Studien an eineiigen Zwillingen, "Zeitschrift für Klinische Medizin", vol. CI, 1924, nos. 1 and 2.

he writes: "What a young student of mathematics (pair 45) says of himself and his twin-brother may be regarded as typical. Both of them (being, by the way, much the most able members of their school form) had up to and including the middle forms, the same reports, that is to say, the same marks, in each subject; when they had got into the higher forms, there were occasional differences between them in different subjects, but the reports taken as a whole were equally good. The strong point in each of them was mathematics, and even within that field they were at first both of them interested in the same problems. For several terms, now, they have both been studying mathematics, but at different universities. That may well explain why, of late, one of them has devoted himself more to geometrical and the other to analytical mathematics. Both of them are very musical, and both play the violin. They like the same composers, and even the same compositions. Neither of them, on the other hand, has any taste for literature, whose study does not to either of them seem worth the pains, so that they do not bother about it."

Noteworthy, too, is the following: "Two male twins aged about 60 years, whom we visited in the village where they lived and whose marked physical resemblance made it obvious that they must have been mon-oval twins, were regarded by their neighbours as eccentrics, and produced the same impression in our own minds. One of the pair, who had remained unmarried, was manifestly more of an eccentric than the other." It is probable that the difference here may have been an expression of the effect of marriage in one instance and bachelorhood in the other.

Handwriting is to some extent an expression of the mental constitution, and Weitz tells us that in mon-oval twins the handwriting may be so much alike as with difficulty to be distinguished. The same remark applies to facial expression and to gestures.

Most of the data obtained by the psychological study of heredity relate to differences in the understanding and in kindred capacities. But differences in temperament and character are, beyond question, no less hereditary, as we learn from daily experience, the only trouble being that these differences are far less easy to determine with scientific precision. H. Hoffmann[1] has done his best to demonstrate their heredity. C. B. Davenport[2] has come to the conclusion that the excitable temperament (as contrasted with the calm temperament) depends upon dominant heredity factors. In one family he was able to trace a proneness to emotional outbursts uninterruptedly through five generations. A cheerful temperament is, according to Davenport, dominant as against a gloomy one, whereas the equable temperament occupies an intermediate position. The same authority believes that dominant heredity factors are at work in the production of that peculiar restless temperament which manifests itself in the wandering impulse (nomadism, fugues), but this temperament displays itself more frequently in males than in females. The results of Davenport's enquiries lead him to believe that proneness to violent fits of temper is the outcome of a certain correlation between epileptic and hysterical disturbances. He finds that the wandering impulse manifests itself chiefly in feeble-minded and psychopathic individuals, for when present in persons endowed with normal powers of judgment it would appear to be usually suppressed.

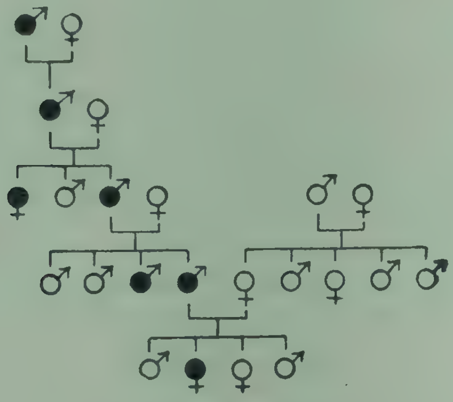

FIG. 169.
Proneness to fits of temper, after Davenport.

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[1] Ueber Temperamentsvererbung, Bergmann, Munich, 1923; Das Problem des Charakteraufbaus, Springer, Berlin, 1926.

[2] The Feebly Inhibited; Nomadism or the Wandering Impulse, with special reference to Heredity; Inheritance of Temperament; Washington, 1915.

The anomalies of character which predispose to crime have already been considered in connexion with the hereditary mental disorders. (See above pp. 459 et seq.)

Very general is a considerable degree of correlation between the qualities of the understanding, the temperament, and the character. Karl Pearson, for instance, has detected a correlation between intelligence and conscientiousness;[1] and E. Webb has found a correlation between a cheerful temperament, on the one hand, and sociability or community sense, on the other.[2] According to Peters, it is the exception rather than the rule for mental qualities to be independent of one another.

R. M. Yerkes has studied the heredity of certain qualities of character in animals.[3] In these animals, "wildness" and "savageness" could be traced in successive generations of various stocks. C. A. Coburn was able to trace the heredity of these same qualities in mice, especially in crossings between tame and wild mice.[4] From out of the mixed offspring, timid and confident, savage and friendly specimens could be bred. For the rest, timidity and savageness did not appear to decline in successive generations, so that there was no evidence of the inheritance of acquired characters. We infer from this that the tameness of our domesticated animals is not the result of the inheritance of acquired characters, but is the result of selection.

The inheritance of particular mental faculties and aptitudes is especially plain in the various kinds of dogs and hounds that are bred for special purposes.

The mental equipment, like the bodily equipment, is, of course, partly dependent upon sex-linked heredity factors. During the discussion of the morbific heredity factors which produce various diseases or anomalies of the central nervous system, we found that a number of them are recessively sex-linked, being therefore referable to defects in the sex chromosome (see above, p. 412). It follows from this that there must be dominant sex-linked heredity factors which are indispensable to the construction of a normal central nervous system, and we may expect that some, at least, of these sex-linked heredity factors must also manifest their working in the mental equipment of those endowed with them.

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[1] The Relationship of Intelligence to Size and Shape of Head and to other Physical and Mental Characters, "Biometrika", vol. V, 1907, p. 105.

[2] Character and Intelligence, Cambridge, 1915.

[3] The Heredity of Savageness and Wildness in Rats, "Journal of Animal Behavior", vol. III, 1913, p. 286.

[4] Heredity of Wildness and Savageness in Mice, "Behavior Monographies", vol. IV, 1922, no. 5.

It has not as yet been possible to furnish direct proof that mental endowment can be sex-linked. In Peters' material we find, indeed, certain indications of this, for he discovered that the resemblance between fathers and daughters was greater than the resemblance between fathers and sons, that the resemblance between mothers and sons was even greater, and that the resemblance between mothers and daughters was greatest of all. But the results obtained by other investigators are not in perfect conformity with these, and it therefore remains possible that the facts observed by Peters may have been the outcome of chance. If we contemplate the resemblances between parents and children while making allowance for differences in sex, we have to bear in mind that here there are two different kinds of interconnexion, which to some extent reinforce one another's working, and to some extent interfere with one another's working. Identity of sex tends to produce increased resemblance between fathers and sons, on the one hand, and between mothers and daughters, on the other; but the sex-linking of some of the heredity factors tends to diminish the resemblance between fathers and sons while increasing the resemblance between fathers and daughters on the one hand and between mothers and sons on the other, seeing that sex-linked heredity factors are never transmitted from father to son. It is a current belief that sons incline to "take after" their mothers, and that daughters incline to "take after" their fathers. Sex-linked heredity would explain such a sequence of events. The scientific study of sex-linked heredity factors tends to show, however, that this current view is no more than an extremely superficial approximation to the true state of affairs. If we suppose the number of haploid chromosomes in human beings to be 24, we should expect only about

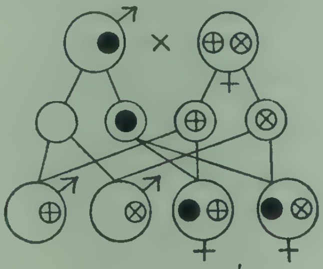

FIG. 170.
Diagram to illustrate the heredity of sex-linked factors.

one twenty-fourth part of the hereditary equipment to be sex-linked.

Long before the beginning of scientific genetics, Schopenhauer advanced and stubbornly maintained the proposition that the "intellect" is inherited exclusively from the mother, and the "character" exclusively from the father.[1] To-day there can be no doubt that the "intelligence" is not a unity which can be transmitted by inheritance as such, for it is the outcome of the working of a great number of heredity factors; and the same is true of the personal character. Still, Schopenhauer did good service in his day by pointing out the preponderant importance of mothers in supplying the hereditary mental equipment of sons, and we have to thank him for adducing a number of examples from history.

In conflict, to some extent, with the foregoing considerations are the observations of Galton, who found that celebrated men had more celebrated relatives in the male than in the female line, and therefore inferred "inherent incapacity in the female line for transmitting" certain "peculiar forms of ability."[2] If there really existed such predominant powers in the male line of inheritance, they would presumably be explicable through the working of hereditary factors localised in what is known as the Y-chromosome (see above, p. 516). Since, however, it is hardly possible to detect talent as such (since we can only recognise it in persons who have been successful or who have become celebrated) we may well suppose that differences in this respect are determined by family tradition, seeing that in "talented families" the offspring in the male line have, more than the offspring in the female line, felt bound to repeat the distinguished achievements of their forefathers —and the males rather than the females will be given by the family special advantages in support of their efforts to do this. Galton himself referred to another possible explanation, pointing out that the female members of distinguished families being inclined to expect more from a husband than do women belonging to ordinary families, less often marry, and have fewer offspring. It is noteworthy, that in the case of men of science and mathematicians (that is to say in domains where more than anywhere else success is due to genuine talent and cannot be achieved simply by glib talkers), Galton found that the maternal line had an exceptionally strong influence. Among 43 mothers of noted representatives of the exact sciences, 8 were actually better endowed scientifically than the fathers. Our general conclusion must, therefore, be that Galton's data do not conflict in any way with the assumption that some of the heredity factors of certain mental endowments may be sex-linked.

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[1] Die Welt als Wille und Vorstellung, vol. II, chapter 43, Erblichkeit der Eigenschaften.

[2] Hereditary Genius, 1914, p. 318.

The differences between the sexes is itself based upon the hereditary equipment. Normally a woman contains all the sex-linked factors twice over, but a man contains only one specimen of each—or, which amounts to the same thing, a woman carries two X-chromosomes, and a man only one. This difference affects all the cells of the organism, and we may presume that unaided it would suffice to account for certain mental differences between the sexes. Superadded are those direct effects of differences in hereditary equipment which are the expression of the hormonal influence of the reproductive glands (the gonads). The varying development of the gonads, thanks to which in one sex they become transformed into ovaries and in the other into testicles, depends upon the above-mentioned differences in hereditary equipment; and once these glands have developed in one direction or in the other, the hormones they respectively produce, masculine or feminine as the case may be, reinforce the bodily and mental distinctions between the sexes—as is clearly proved by experiments in castration and transplantation. Still, we may presume that in human beings, as we know to be the case in animals, the differences between the sexes are not exclusively dependent upon the differences in hormonal influence. A unilateral bullfinch hermaphrodite described by H. Poll showed on one side of the breast the bright-red feathers of the cock bird and on the other side the grey feathers of the hen.[1] The respective areas were sharply delimited along the middle line of the body. Several other cases of the kind have been recorded. Here the hereditary equipment of the cells of the respective sides of the body must have determined the differences, for obviously the hormones produced by the gonads, operating through the blood circulation, must have had the same effect on both sides of the body.

Men are specially selected for the control of nature, for success in war and the chase and in the winning of women;

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[1] Zur Lehre von den sekundären Geschlechtscharakteren, "Sitzungsberichte der Gesellschaft naturforschender Freunde", vol. VI, Berlin, 1909.

whereas women are specially selected as breeders and rearers of children and as persons who are successful in attracting the male (such powers of attraction being more often exercised instinctively than consciously, and frequently taking the form of coyness and apparent reluctance). Hence arise the essential differences between the sexes, differences which are apt to be misinterpreted under the spell of individual valuations. Now man, now again woman, is said to be fundamentally ill-tempered or immoral; one day we are told that the eternal feminine, another day that the eternal masculine, allures us. This much, at any rate, is certain, that there are essential differences between the sexes. Not only do these differences exist, but they are natural and normal.

In his book on the inheritance of acquired characters, Peters describes the empirical differences between the sexes as follows: "But in the qualities connected with activity and emotionality woman shows herself superior to man. In woman, likewise, the emotional after-effects of experiences are stronger. Woman, nevertheless, is less often cantankerous and morose; less frequently ironical, less inclined to sexual debauchery, less covetous; but she is more often tender towards children, more religiously inclined, more indifferent to politics, more easily influenced in her opinions, less inclined to stick to the matter in hand, less courageous, and more prone to be ruled by convention. In general dexterity she excels man, but she is inferior to him in respect of mathematical and literary endowments. On these matters consult Heymans."[1]

In intelligence tests, girls on the average shaped quite as well as boys of the same age. In this connexion, however, we must remember that girls are usually in advance of boys as regards bodily and mental development. In the latter half of the second decade of life, when this difference between the sexes is greatest, it amounts to several years. Nevertheless, among the gifted children studied by L. M. Terman,[2] boys were in a majority from the first, and the excess of boys over girls among gifted children increased during the later years of adolescence. It would seem, then, that a girl's power of mental achievement attains its climax earlier than a boy's. Still, even during the period of university studies, the women students are on the average at least as successful as the men students—and they certainly do better in examinations. The remarkably brilliant achievements of many women students would lead us to expect that in subsequent life they would do great things, but such expectations are hardly ever fulfilled. "Great women" endowed with "greatness" in the sense of outstanding creative faculty are practically unknown. Especially rare are high mathematical capabilities in women; and the same is true of a talent for chess, a game in which skill certainly affords a measure, through a one-sided one, of intellectual powers.

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[1] G. Heymans, Psychologie der Frauen, second edition, Heidelberg, 1924.

[2] Genetic Studies of Genius, Stanford University Press, vol. I, 1925.

In perceptive powers and in memory, woman is at least equal to man, but on the average she falls behind him in power of imagination and in critical faculty. Of course there are many women who excel average men in these matters. If, nevertheless, the independent achievements of women in the fields of science and art lag behind those of men, this is manifestly due to the fact that women have other interests than men and other impulsive trends. These differences between the impulsive life in the respective sexes are much greater than differences in the matter of intellectual endowment—and this applies, not only in the domain of the sexual impulses, but in the food-seeking impulses as well, and still more in the social impulses. Determination by wishes is, normally, more powerful in women than in men. Their impulse to self-assertion is not stronger than that of men but is directed along different lines. A woman wishes, above all, to be regarded as beautiful and desirable, whereas a man wants to be regarded as a hero and as a person who gets things done. Man has more courage than woman in attack, whereas woman shows more valiancy in suffering. Since women are selected by nature mainly for the breeding of children and for the allurement of men, their interests are dependent upon those of men and of children, and are directed towards persons rather than towards things. Owing to the particular nature of her part in life, woman is endowed with more imaginative insight, more empathy, than man. She can more readily put herself in another's place, she lives more for others, her main motive being her love for her husband and her children—and her desire to foster illusions in her husband's mind. Man lives rather for himself, his main motive being selfishness and material interest. Speaking generally, a love of knowledge for its own sake and an inclination to seek non-personal ends are rare in men also.

We are often told that woman does everything under stress of erotic motives, but this assertion is based upon an excessive and misleading extension of the notion of the erotic. If we are going to say that all our impulses are erotically tinged, the word ceases to have any meaning. Weininger, who detested women, could see in woman, side by side with the mother, nothing but the whore—caricaturing equally the mother type and the whore type.[1] The woman of the motherly type is not so keen on having children in general as on bearing children to the man of her choice, whom she must be able to respect and trust; and the true prostitute is not one who lures man impulsively as does the "whore" of whom Weininger is speaking, but only one who wishes to attract him in order to earn money by the sale of her person. Still, though Weininger exaggerates grievously, there is an element of truth in his typology, inasmuch as the specifically feminine impulses are, on the one hand the impulse to mother people and to care for them, and on the other the impulse to allure the male. In one woman one of these impulses may predominate, and in another woman the other; and according as a woman's life history varies, one or other of the impulses may be encouraged or inhibited. Assuredly it is irrational to reproach a woman for being endowed with either or both of the impulses in question. Nothing can be more absurd than to speak of one sex as being intellectually or morally "higher" or "lower" than the other. Each sex has its own peculiar tasks to fulfil in the life of the race, and must be fitted for these tasks by the inheritance of particular bodily and mental characters.

Differences in intellectual endowment are to some extent shown forth in bodily characteristics. In this respect I may refer once more to Kretschmer's studies concerning physique and character (see above, p. 462). Every one knows that the larger kinds of dog are inclined to be slow and deliberate in their movements, and that smaller dogs, such as terriers, are livelier and fussier in their ways. We see corresponding differences between large human beings and small ones. The caricaturist loves to depict an idealist as slender and pale, an opportunist as stout and sanguine. I have no doubt that if ten musical persons were to be placed in one group and ten unmusical persons in another, I should know without enquiry which was which, though I should probably not be able to give reasons for my judgment; and I am sure that any one of my readers could draw the same distinction. In part, no doubt, this is because the consequences of mental activity or inactivity, and the kind of mental activity, stamp themselves upon the countenance, and it is from this imprint that we infer a person's mental constitution. That is how we distinguish a cultured person from an uncultured one, even though the cultured person may be much less gifted hereditarily. To some extent, however, the relationships between mental endowments and bodily appearance are obviously more direct than this. A person whose mental hereditary equipment is a good one can be recognised with great probability when his forehead, his nose, etc., are of a particular shape. That is the main explanation of the great interest we all feel in the examination of the portraits of celebrated persons. The bodily peculiarities in question and their relationship to mental endowments depend, in part, upon the effects of the internal secretions, for these, as Kretschmer has pointed out, have effects both on mind and on body. Although we must be careful not to extend this notion of internal secretions too widely, there can be no doubt that there are other connexions at work here.

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[1] O. Weininger, Geschlecht und Charakter, twenty-fifth edition, Leipzig, 1924; English translation, Sex and Character, Heinemann, London, 1906.

To a certain extent intellectual gifts are evidenced by the size of the head, which affords some indication of the size of the brain. We know that where the body is alone concerned, a well-formed organ is as a rule more effective functionally than one which is less well developed. It does not, of course, follow that an exceptionally large organ is always better functionally than a smaller one. Persons with a large chest measurement are not invariably more vigorous than persons with a small one. In exceptional instances, the functional capacity may be very small notwithstanding a large chest measurement, as we see, for instance, when there is a barrel-shaped chest with pulmonary emphysema. On the average, however, persons with a large chest measurement are more efficient functionally than persons with a small chest measurement; and in the case of those whose chest measurement falls below a certain figure, we can be confident that their bodily efficiency will be below par. Similar considerations apply to the brain. The simplest rough-and-ready way of ascertaining the size of the head is to measure its circumference. According to the comprehensive researches of Bayerthal,[1] when the circumference of the head is less than 52 cm. we hardly ever find that there are any noteworthy mental achievements, and when the circumference of the head falls below 50·5 cm. the intelligence is no longer normal. Genius is out of the question in persons the circumference of whose head is less than 56 cm. The well-known psychiatrist Ziehen[2] puts the limit below which feeble-mindedness is to be suspected somewhat higher, namely, at 52 cm. K. Röse,[3] examining a very large number of school-children in Dresden, found that, on the average, their heads were smaller in proportion to the badness of their reports. In the higher schools (gymnasia) of Germany, it was found that the abiturients with the best reports had, on the average, the largest heads, although they were younger on the average than the abiturients with less satisfactory reports. In Strasburg, again, the anatomist and anthropologist W. Pfitzner[4] came to the following conclusion, as the result of his socio-anthropological studies: "A higher degree of intelligence is plainly indicated by higher average stature and by a correspondingly larger average increase in the size of the cranial portion of the head." Even R. Martin,[5] an extremely cautious anthropologist, declares that "mental functioning is to a certain extent dependent upon the weight of the brain".

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[1] Ueber den gegenwärtigen Stand der Frage nach den Beziehungen zwischen Hirngrösse und Intelligenz, "Archiv für Rassen- und Gesellschaftsbiologie", 1911.

[2] Die Erkennung des angeborenen Schwachsinns, "Zeitschrift für Schulgesundheitspflege", 1907.

[3] Beiträge zur europäischen Rassenkunde, "Archiv für Rassen- und Gesellschaftsbiologie", 1905 and 1906.

[4] Sozialanthropologische Studien, "Zeitschrift für Morphologie", 1899–1903.

[5] Lehrbuch der Anthropologie, Jena, 1914, p. 651.

In the paper referred to on page 594, Karl Pearson reports that in Cambridge graduates he found a comparatively small correlation between intellectual achievement and length or breadth of the head ($r = 0{\cdot}11$ or $0{\cdot}10$). It must be remembered, however, that here we have to do with a very rigorously selected group. In large populations, the correlation will certainly be found to be much higher.

It might be expected that an exceptionally large brain would be needed for the storing-up of an exceptional amount of knowledge, but this seems not to be the case. Sometimes persons with an astonishingly good memory will be found to have very small heads; and such one-sided talents as the musical and even the mathematical would appear to be compatible with smallness of the head; but more comprehensive mental endowments and a well-developed general power of judgment are not so compatible.

A. Woods[1] has found that the majority of great men have a large or a long nose, whereas short noses are hardly ever found in them. Highly gifted groups of persons have, on the average, longer noses than less gifted groups.

It is a remarkable fact that certain persons are persistently inclined to deny the inheritance of mental endowment although they do not find it possible to deny the inheritance of bodily qualities. This is because they believe the mind (or the soul) to have a very different origin from the body, an origin which is supposed to have nothing to do with biology and heredity. It must, however, be regarded as incontestable that mental qualities are no less directly inheritable than bodily. There must be some sort of interconnexion between body and mind, although the problem concerning the nature of the interconnexion may be left open. The materialist conception, the idea that mental processes are exclusively the outcome of bodily activities, is neither presupposed nor supported by the doctrine of the inheritance of mental qualities. The unwarranted fear that an admission of the inheritance of mental qualities will lend support to materialism has led people to maintain that not the mind as such is inheritable but only its bodily instrument. It has been contended, for instance, that the mind of a deaf-mute is simply a mind which has been deprived of the use of the bodily organ of hearing; the mind of a blind person, a mind which has been deprived of the use of the bodily organ of vision; the mind of an idiot, a mind which has been deprived of the use of certain parts of the brain; and so on. It remains questionable, however, whether the mentality which is left after the abstraction of all these capacities can still be regarded as in any way individualised, as equipped with a distinctive quality of its own, or whether it must not rather be considered identical with the mental or spiritual principle of the world in general. The underlying substance of the body, considered generally, is not looked upon as individualised, the view being rather that this substance is one and the same amid all changes in the individual body; and this substance, of course, is not inheritable per se, although it is the foundation which renders all heredity possible. Similar considerations may perhaps apply to the mental world. The substance which underlies the individual configuration of the mental, would in that case not have to be regarded as heritable, or as individualised at all; but the individual mind would, like the individual body, still be rooted in the hereditary equipment.

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[1] What is there in Physiognomy? I. The Size of the Nose, "Journal of Heredity", vol. XII, 1921, no. 7.

Even more important, perhaps, than a recognition that mental qualities are inherited is the recognition of the fact that nothing out of all that a human being acquires during his lifetime through practice and experience can be transmitted by inheritance. A person hereditarily endowed with a certain amount of musical talent may learn to play the piano or to engage in some other musical activity, but this will not make the musical endowments of his offspring greater or less than they would have been. What a human being or any other living creature inherits is a sum total of possibilities of reacting (see above, p. 33). Which of these possibilities of reacting are realised, and the way in which they are realised, during the lifetime of the individual, has no bearing whatever upon the hereditary equipment of that individual's offspring. When a possibility of reacting is absent, of course it cannot be transmitted by inheritance. In days of old, people sometimes endeavoured to teach the deaf to hear by making loud noises in their presence. To-day this seems to us absurd. The deaf cannot be made to hear by practice, or by punishment. No amount of exercise in colour vision can give the lacking colour sense to the colour-blind, whose capacity for colour vision will remain what it is as the outcome of hereditary equipment. If practice and education are to have any effect, the requisite hereditary foundation must be present, and then, within certain limits, a development of faculty is possible. The results of education depend mainly upon the accumulation of a store of memories and upon habituation to certain views and to certain modes of behaviour. Memory and mental plasticity in general are greatest in youth, so that youth is the period during which the individual human being obtains knowledge and adopts views and habits appropriate to the life of the society to which he belongs. In the mental domain, as in the physical, there occur permanent modifications which usually persist in the individual throughout his lifetime, are transmitted by way of tradition to the next generation, and may thus continue through countless generations. Speech and religion, for instance, are such permanent modifications. Lamarckism, too, the doctrine of the inheritance of acquired characters, is itself one of these permanent modifications. A convinced Lamarckian remains a Lamarckian at heart notwithstanding all the evidence brought forward against his belief. With very few exceptions, such persons remain Lamarckians to the end of their days.[1] "Habit is second nature", says the proverb. What is acquired in youth becomes "second nature"; but the "first nature", the nature grounded upon the hereditary equipment, remains unaffected by this "second nature". Consequently, the educational influences which have been at work on past generations, can have had no effect upon the hereditary equipment of the present generation; and educational influences at work upon the members of the present generation, can have no effect upon the hereditary equipment of their offspring in future generations. Goethe's lines

> What thou from thy forefathers hast
> Make it thy heritage, thine own,

apply only to external goods, to the acquirements of civilisation as handed down by tradition; they do not apply to those goods which are truly a heritage in the biological sense. The mental goods which are truly inherited from our forefathers have not to be "made our heritage", have not to be "made our own", by us as children; and whatever the forefathers acquired through inheritance must be inherited by the offspring before these can possess it.

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[1] It would seem that the various human stocks are variously predisposed to Lamarckism.

In his work on the inheritance of mental qualities (see above, p. 591), the psychologist Peters writes: "When Johannsen says that hitherto experimental research has not been able to bring forward a single instance of the inheritance of acquired qualities, this proposition applies with full force likewise to the inheritance of mental qualities." Here it makes no difference whether a quality or a faculty be acquired "actively" or "passively". There is no ground for a distinction between passive and active acquisitions. Again: "The so-called active acquisitions of modes of behaviour, qualities, and capacities are dependent upon external stimuli, that is to say upon the environment."[1]

If Pavloff,[2] having trained mice to come to their food at the sound of a bell, found that the period of training could be reduced in successive generations, all that we need infer from this is that Pavloff himself gradually became more skilful in the training of mice.

If it is found that the children of cultured parents are on the average more gifted than the children of uncultured parents, this is not because the parents of these gifted children acquired culture, but because the parents already possessed the hereditary equipment inclining them to make themselves cultured. Educability, or the capacity for culture, is inheritable. On the other hand, it is a familiar experience that the offspring of cultured families are, often enough, conspicuously untalented persons, and are persons who, despite the best cultural and educational facilities, prove quite unable to assimilate the elements of a higher culture. Such facts tell strongly against the view that the acquirements of culture are inheritable, but they are perfectly comprehensible in the light of the basic mendelian law of heredity. Consequently there is absolutely no prospect that we shall ever be able to bring about a permanent elevation of the human race by means of education and practice. This is an all-important principle of racial hygiene.

[1] Op. cit., p. 307.

[2] Reported in the American periodical "Science", 1923.

Those who find it depressing to be told that none of the acquirements which are the outcome of our persistent endeavours towards perfection can enter into the hereditary equipment of our offspring, may be reminded that were it otherwise the coming generation would be burdened, not only with the beneficial acquirements but also with all the dross, with all the errors and absurdities, all the mean and despicable acquirements and practices of the present and past. In so far as these undesirable elements of our civilisation are not the outcome of the hereditary disposition of our contemporaries, but result merely from the circumstances of the time, our offspring will not be hereditarily burdened with them. The coming generation will be able to advance towards greater heights in proportion to the extent to which we ensure that they shall be born out of good stocks. Everything turns on this. The destiny of our race and civilisation depends upon whether contemporary human beings prove intelligent enough to rid their minds of the dominant prejudices concerning the omnipotence of environment, and to effect the necessary transformations in our outlooks on life and in all our institutions.

Among the geneticists who are to be taken seriously there now remain scarcely any supporters of the Lamarckian theory, but Lamarckian views are still enthusiastically advocated by journalists and others who cater for the popular taste. Ever since the days of Rousseau and his disciples, Lamarckism has been an article of popular faith. The biologist Paul Kammerer[1] gives expression to a widely held conviction when he says: "Human progress stands or falls with the inheritance of acquired characters." The popular writer A. Elster[2] expresses a similar view. In such instances, biological progress is usually confounded with the progress of civilisation. The achievements of civilisation, however, are transmitted from generation to generation, not by biological inheritance, but by tradition (mainly, nowadays, through the instrumentality of print). Here there is no question of the inheritance of acquired characters. But biological progress, in the sense of an increase in the capacities of the human race, is not in any way determined by the inheritance of acquired characters or qualities. Elster is utterly mistaken when he says that, without the inheritance of acquired characters, "there can be no question of an influencing of the biological environment and education"; and that unless acquired qualities are inherited, all the items of the present human hereditary equipment must have existed just as they are for millions of years, "must already have been present in pithecanthropus or some such creature". No one wants to deny the effects of environment and education. What we say is, that these effects are confined to the individual. The opponents of Lamarckism do not deny the transformation of species, but they say that this transformation is the outcome of the cooperation of hereditary modification and selection. There are no solid grounds for the belief that there is a natural law of evolution towards higher things, as Elster believes, and as Oskar Hertwig believes. If we are to prophesy a further transformation of the human race, we must prophesy with caution, not upon the ground of such preconceived opinions, but only upon the ground of the facts of experience. These facts, which will be thoroughly discussed in the sequel to the present work, force us to the conclusion that at the present time as regards mental functional capacity the human race is on the down grade. Those who give expression to such a view are usually charged with "pessimism", as if to accuse them of this were to refute their arguments. In actual fact, the science of genetics is neither "pessimistic" nor "reactionary", for, on the contrary, it shows us how we can put a stop to the retrogressive movement and change it into an advance. The Lamarckian blindness to obvious facts, on the other hand, is beyond question reactionary.

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[1] Neuvererbung oder Vererbung erworbener Eigenschaften, Seifert, Stuttgart, 1925. (Regarding Kammerer, see below, footnote[2] pp. 674–5.

[2] Sozialbiologie, Gruyter, Berlin and Leipzig, 1923.

CHAPTER FOURTEEN

TALENT AND PSYCHOPATHY

MARKED deviations from the average are usually morbid, and are invariably to be regarded as morbid in those cases where the nature of the deviation is such as to determine a reduction of the chances of self-preservation to a point below the mean. Exceptional gifts often lead to (or are at any rate associated with) such a reduction. Many talented persons are incompetent to cope with the demands of everyday life, demands which average persons endowed with an ordinary share of healthy common-sense can fulfil without difficulty. What applies here to individual self-preservation, applies still more to the preservation of the family. A considerable proportion of talented persons remain unmarried. We find especially often that geniuses are single; or, that if they do marry their family circumstances are unhappy because the powers of the genius are so apt to fail when it is a question of the give and take essential to a happy connubial life.[1] The usual result is that the family dies out. Now, when a person's mental constitution is such as to interfere with the chances of self-preservation or family preservation, we say that such a person is psychopathic, or, in extreme degrees, insane. It therefore becomes essential to discuss the question as to how far genius or exceptional talent is morbid, all the more seeing that the problem is one which concerns the practical attitude of racial hygienists towards psychopaths.

If we are to consider this matter without prejudice, we must first of all draw a sharp distinction between a scientific contemplation of phenomena and a contemplation which involves judgments of value. Illness or disease is a scientific concept, and this concept is one which does not necessarily involve a negative judgment of value. It is possible that certain morbid conditions may be desirable, while remaining morbid. If we find it necessary to decide that the mental life of a particular man of genius is morbid, this does not per se detract from the value of his achievements. Morbidity is not synonymous with inferiority. On the other hand, the coincidence of exceptional achievement with morbid characteristics of the mental life is too common to be explained as the outcome of mere chance. No doubt psychopathy is very common, genius apart; but it is commoner in celebrated persons than in the general run of the population. Even the three men who would generally be signalised as the three greatest Germans of modern times—Kant, Goethe, and Bismarck—were not free from psychopathic taint. Many count Luther among the greatest of all Germans, and he was markedly psychopathic. So conspicuous is this association, that we are actually compelled to ask ourselves whether genius be at all compatible with complete mental health. I cannot myself call to mind any person of altogether outstanding distinction in whom the discerning eye of the psychiatrist would fail to discover a morbid anomaly—which, of course, does not prove that no such persons have ever existed. Geneticists would be greatly indebted to any alienist who should seek out and report on such exceptional instances.

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[1] This matter is ably treated by H. Kurella in *Die Intellektuellen und die Gesellschaft*, Bergmann, Wiesbaden, 1913, p. 73.

The catchword "genius and insanity" was introduced by Lombroso, who was the first to discuss this problem at any length. The phrase has caught on, but it is misleading, seeing that the creative activities of the genius cannot possibly be undertaken by any one suffering from genuine insanity, especially from the insanity which arises from the actual destruction of brain tissue. If, for instance, dementia ensues in the course of schizophrenia, the mental functions are very gravely impaired. On the other hand it would seem that the schizoid phases which precede the development of schizophrenia may, under certain conditions, fan the fires of the spirit. To give instances, this apparently happened in the cases of Hölderlin, Scheffel, and Strindberg. Ampère probably passed through a phase of schizophrenic mental disturbance (Kretschmer is our authority here); and so did Newton. Dementia praecox may, in a sense, be compared to cicatrisation after an inflammation: and in the early period of mental inflammability or vulnerability altogether exceptional mental achievements may become possible. This is the way in which Kretschmer looks at the matter, if I understand him aright.

Schizoid psychopathy often leads to a one-sided and lasting concentration of interest upon things to which most persons are comparatively indifferent. Obviously such a concentration may notably contribute to discoveries and inventions, and the records concerning a good many scientists and investigators sustain such a hypothesis. The ordinary man is wont to be fairly well satisfied with the persons and things which comprise his environment; it is an environment in which he feels at home; but schizoid psychopaths, especially when they are persons of great gifts, are apt to be extremely dissatisfied with the inadequacy of ordinary persons and ordinary things. That is why they are born "reformers". The great commander and the great statesman are sometimes called upon, for the furtherance of their tasks, to sacrifice thousands or hundreds of thousands of their fellow men, and a normal being would shrink from such an obligation. Here the activities of the man of genius are facilitated by a touch of "moral insanity" such as is often found in schizoid psychopaths, its main foundation being a lack of sympathy with the woes of others. "Utter indifference as to the fate of the individual, in conjunction with an inclination for the systematic, rigid, and logical application of principles, and for unqualified justice (these may all be expressions of the schizoid taint), can, as well-compensated elements of the personality, produce men having energies hard as steel and an invincible tenacity." Kretschmer gives Frederick the Great as an instance. The philosopher again, when circumstances demand from him a revaluation of customary values, must never allow his sympathy with individuals to hold him back from his task. Even Kant was mainly regarded by his contemporaries as the great destroyer. On the other hand, self-sacrifice on behalf of impersonal ideals is regarded by Kretschmer as characteristic of certain schizoids. Much that has been said of the schizoids applies also to their close kin the paranoid psychopaths. It need hardly be said, however, that psychopathic predisposition is not synonymous with exceptional talent. What I am attempting to show is that when the other factors of exceptional mentality are present, the psychopathic taint may contribute to remarkable achievements. In the book market there are published year after year numerous works which, in their eccentricity, their meaningless systemisation, and their empty formalism bear the unmistakable stamp of schizoid or schizophrenic psychopathy. Most of these unsystematic systems (if I may use the phrase) are of no importance whatever, and yet there are not a few important writings which exhibit kindred characteristics. At this very time, schizoid products are the mode in literature and art. More especially do the painting and the other plastic arts of the contemporary world exhibit, in great measure, schizoid characteristics.[1] Here, of course, we are entering a domain with which real talent has little concern.

Speaking generally, there is no doubt that the existence of epilepsy cannot further brilliant achievement. I suspect, therefore, that in the case of those great men who are reported to have been epileptic (such as Julius Caesar, Paul af Tarsus, Mohammed, Francis of Assisi, Alfred the Great, Peter the Great, Napoleon I, and Dostoeffsky), there was merely an accidental coincidence of epilepsy with genius; or else, which is more probable, that the morbid symptoms in these distinguished persons were not those of true epilepsy, but only epileptoid manifestations of a hysterical temperament. It is obvious that in earlier days hysteria was often confounded with epilepsy, the two being lumped together as "morbus sacer". We are indebted to A. Grotjahn for a collection of the information relating to noted epileptics.[2]

The *hysterical temperament* is naturally more important in relation to high-grade mental activity than is any other mental anomaly. Concerning this matter, the psychiatrist K. Birnbaum writes:[3] "Hysteropathic happenings permeate the mental life of all civilisation and all epochs. . . . Hysteropathic manifestations arise by preference upon the foundation of a peculiar and abnormal psychical constitution. . . . The effects of this constitution greatly transcend what is otherwise possible to the human will and the human abilities in the field of mental life." Morbid traits which seem to me referable to the hysterical temperament were exhibited by Paul of Tarsus,

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[1] Cf. H. Prinzhorn, Bildnerei der Geisteskranken, second edition, Springer, Berlin, 1923.

[2] Soziale Pathologie, third edition, Springer, Berlin, 1923, pp. 298 et seq.

[3] Psychopathologische Dokumente, Springer, Berlin, 1920.

Mohammed, Luther, Pascal, Rousseau, Frederick the Great, Napoleon I, Blücher, Goethe, Schopenhauer, Wagner, Nietzsche, and Tolstoy. In the field of religion, art, and politics, we find that an abnormal susceptibility to determination by wishes may contribute to great achievements, especially when there is a strong impulse to self-assertion, such as is usually found in persons of the hysterical temperament. Even in normal human beings, determinability by wishes is one of the foundations of faith, love, and hope. It is natural, therefore, that an abnormally high determinability by wishes should be a contributory cause of exceptional achievement. It can make goals seem attainable which the sober reason would regard as hopelessly out of reach. "The word impossible has no meaning for me", said Napoleon. This is the source of the faith that moves mountains, of the faith which pays no heed to the testimony of the senses and of reason, and thus facilitates the performance of the "impossible". Furthermore, worldly success depends in great measure upon exercising an effect upon the masses, and this effect is mainly produced by the contagious spread of brilliant wish illusions. A great proportion of the "strong men" of history have been persons with a hysterical temperament. Kretschmer has adduced convincing evidence to show that the hysterical "tetanisation" of the unconscious will may give the impression of immense will power.[1] Leading statesmen and military commanders must be predisposed to such an occasional tetanisation of the will—which, however, involves the danger that they continue to strive blindly towards a goal long after the goal has ceased to be worth attaining.

In the mental domain, likewise, such a predisposition may contribute to notable achievements. Fechner, who was once affected for a month by a severe hysterical illness accompanied by photophobia, reports of himself: "If any object came into my ken my thoughts would begin to circle round it, returning to it again and again, so that these thoughts went on boring, as it were, into my brain." Here obviously, we have one of the prerequisites for discovery. In like manner, Rousseau writes: "I cared not a jot for the rest of the world, apart from the objects with which I was directly concerned."[1] The spirit that animates great poets and artists is akin to, and perhaps identical with, the spirit that possesses the hypobulic.[2] The distressing intenal tension that proceeds from the impulses which have been repressed into the subconscious may be a source of great achievement; and the tension may be relieved by expression and configuration, in which the subconscious becomes conscious.

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[1] Ueber Hysterie, Thieme, Leipzig, 1923.

And when the man was, in his torment, mute,
A god then gave me words to voice my pain,

Goethe puts into the mouth of his Tasso. Heine says of himself:

Illness is but the reason sore
Why to create we strive amain;
Creating, I could health regain,
Creating I was well once more.

Nietzsche tells us the same thing. We must further take into account that the elementary need for self-assertion which usually dominates hysterically predisposed persons impels many poets and other artists to words and actions which produce a strong impression upon their contemporaries and upon posterity. To such apply Schiller's words:

For of all life's greatest gifts
Fame is surely the highest good.

Socrates long ago recognised the power of this inner spirit, the power of the daimon. Plato says of it, speaking of himself and others: "If anyone lacking the madness of the muses should try to enter the gate of poesy, his poems will pale before the art of one affected by the creative madness." Aristotle, too, referred to the morbid predisposition of great writers, artists, philosophers, and statesmen.

The figures staged by Ibsen as persons of note were almost all hysterics. Peer Gynt is the archetype of a hysteric and he has a great success in the world. Don Quixote is almost entirely composed of the impulse to self-assertion and of determinability by wishes. Nietzsche "felt himself, in a disquieting way, to be somehow akin to the almost mythical figure of Don Quixote".[1] In his youth, when he fancies that this famous work of Cervantes was an attack upon romanticism and idealism, he opined: "One of the most harmful of all books is Don Quixote." At a later date, however, Nietzsche wrote: "If we ever become fully aware of the quixotism of our feeling for power and become fully awake—then we humble ourselves like Don Quixote—a terrible end."

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[1] Quoted by Birnbaum.

[2] "Hypobulia" denotes infirmity of will, and Kretschmer classifies as hypobulic, hysterically predisposed persons in whom the subconscious impulsive life plays a dominant part

It lies in the nature of the case that in the scientific field the hysterical temperament should more often lead to imaginary than to genuine discoveries. Hysterically predisposed investigators are only too apt to regard brilliant flashes of insight as definitive truths. Especially when criticised by rivals and when defending themselves against attack do such investigators become more and more obstinate, until at last they stake their whole scientific personality upon their contentions.[2] Experience shows, moreover, that such a stubbornly advocated proposition may sometimes be a true one. Consequently, it cannot be said that the hysterical temperament is necessarily and at all times unfavourable even to scientific discovery. It is true that the general upshot of the activity of hysterical authors is to darken counsel. To this we must attribute the present flood tide of magian superstition. If there is a hysterical flight into illness, there is also a hysterical flight into obscurity. Persons who dread the light of the understanding take refuge in the magian chiaroscuro of animistic and occultist doctrines. Scientists who are not equal to the occasion, who are not sufficiently well equipped to meet the demands of modern research, but whose desire for self-assertion makes it impossible for them to admit the fact to themselves or others, throw the handle after the hatchet and solemnly announce that science has left us in the lurch and that magic alone can bring salvation. Thousands upon thousands of reputedly "cultured" persons, who find science beyond their reach as the fox found the grapes, seek spiritual nutriment from such writings. Persons who have taken to journalism after failing in a scientific career, fill the columns of great newspapers with pseudo-scientific balderdash. Unfortunately the hysterical mimicry of genuine talent appears to thrive above all in Germany, whereas the comparatively sober Anglo-Saxons are more level-headed in such matters. The German public is far too readily convinced that a flux of big words is an indication of "profundity". It would seem to be the doom of this "people of thinkers" that from time to time such magians should be mistaken for great philosophers. Fortunately there are still among us quite a number of persons possessed of sound common sense, who feel that the convulsions of the magians are exuberant.

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[1] See K. Hildebrandt, Wagner und Nietzsche, Hirt, Breslau, 1924, p. 253.

[2] H. G. Wells' short story, The Moth, is a brilliant imaginative study of such a type.—TRANSLATORS' NOTE.

Even so great a genius as Schopenhauer was not entirely free from hysterical taint. He experienced in his own person the power of subconscious impulses, of the "will" as he called it, as contrasted with the power of conscious insight, the power of the "intellect"; but the "primacy of the will over the intellect" does not extend so far as he supposed. For Schopenhauer, the whole world was a hysterical wish illusion. I do not mean that he actually used these words, but that such was the general significance of his doctrine. According to Schopenhauer, the world is an illusion of the "will to live", whose foundation he sees in sexual desire; and since, for him, all will is tinged with suffering, he regards the world as an efflux of hysterical passion. This being so, deliverance is necessary and possible. If the phenomenal world is a painful product of the "will", and as such has no real existence, then, according to Schopenhauer, the world must cease to be as soon as it is recognised to be an illusion. Thus Schopenhauer's doctrine of deliverance is a forerunner of Freud's "psychoanalysis" (see above, p. 453). When an illness is recognised by the sufferer as unreal, as the product of subconscious desire (repressed affects), then, according to Freud, it vanishes. He teaches regarding individual cases of hysterical illness precisely what Schopenhauer taught regarding the world in general; or, conversely, for Schopenhauer the world was a hysterical wish illusion. Since, however, notwithstanding this "recognition" of Schopenhauer's, the world has continued to exist, we may venture to doubt whether the doctrine of determination by wishes is really applicable in its case.

It is manifest, that in the last resort, Schopenhauer did not take his own doctrine seriously. In the subconscious, he, like every one else, was convinced of the reality of other persons and things. Had it been otherwise, why should he have emptied the vials of his wrath on Hegel? Had Hegel been nothing more than an idea of Schopenhauer's, Schopenhauer would merely have had to "recognise" that the phenomenon Hegel had no real existence and the phenomenon Hegel would have ceased to exist. His wrath against Hegel, and his general behaviour throughout life, showed that his practical estimate of reality differed from his doctrinal estimate. He was, himself, affected by a "primacy of the will over the intellect"; that is to say, he suffered from an undue inclination to determination by wishes, which led, in his general attitude towards the universe, to a typically hysterical splitting of consciousness. His powerful self-assertive impulse led him to believe that he was the "one and only heir to the throne of Kant", that he had grasped the "principium individuationis" and had been the first to understand fully the nature of the "thing in itself" which inhered in the "will". But his doctrines would never have secured so extensive a vogue had they not given expression to a very important constituent of the human mind, namely, determinability by wishes. Nevertheless, we must recognise the limitations of Schopenhauer's philosophy. "Idealism" (of which Schopenhauer was an extreme advocate) teaches that the mind makes the body. This is an absurd doctrine, and in current idealism there is a large admixture of wish illusion.

The mental anomalies which are classed under the head of *psychasthenia* (or neurasthenia) seem likewise, in many instances, to contribute to brilliant achievements. Charles Darwin was a psychasthenic able to work only for a few hours every day, and yet he contributed more to human knowledge than a thousand other investigators put together. Similar remarks apply to Fechner and to Herbert Spencer. Conrad Ferdinand Meyer was also extremely psychasthenic. Nay, even Bismarck, though few may have suspected it, must be classed among the psychasthenics. Almost all his life, Bismarck suffered from nervous troubles, and, despite his giant frame, he was, as Birnbaum says, endowed with unduly delicate nervous susceptibilities. It need hardly be said that, generally speaking, psychasthenic taint is a hindrance rather than a help to mental achievement. Mörike, a sufferer from high-grade psychasthenia, was unable to pursue his profession.[1] Nietzsche, even after relinquishing his professional duties, suffered much from undue liability to exhaustion and from sleeplessness. That was why most of his great works remained fragmentary, so that he was never able to complete the finished wholes appropriate to his high gifts in other respects. Darwin, happily, was a man of means and leisure. Apart from his psychasthenia his hereditary constitution was a good one, and he was thus enabled to do his epoch-making work.

The brain of the genius is almost always busily at work, and if it more frequently exhibits signs of exhaustion than do the brains of ordinary mortals, we must not infer that this necessarily depends upon an abnormal liability to fatigue such as is characteristic of psychasthenia. "My mind is as restless as an everlasting firework", writes Goethe of himself. Hebbel says much the same thing: "I am at all times what most people are when suffering from fever." The average brain would probably go on strike after doing only a small fraction of the work done by the brain of the genius; and if the average brain is usually spared the symptoms of psychasthenia, this may well be because it does not usually perform even so much as this fraction. When, however, the abnormally vigorous activity of the brain of the genius suffices of itself to impair health, we must look upon its activity as morbid.

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[1] A. Müller, Bismarck, Nietzsche, Scheffel, Mörike, Marcus and Weber, Bonn.

The *manic-melancholic taint* would also seem able to contribute to brilliant achievement. The mental disturbances from which Conrad Ferdinand Meyer, Anselm Feuerbach, and Robert Mayer, suffered come within the group of manic-melancholic disorders as usually defined. Byron, Kleist, Schopenhauer, and Grillparzer were of melancholic temperament, but in Schopenhauer the paranoid-hysterical taint was more influential. A powerful impulse to rid the world of its evils does not usually arise in persons who are well content with themselves. "The cultivation of suffering, of great suffering—do you not know that this and nothing but this has hitherto led to all the advancement of mankind?" Thus writes Nietzsche. There can be no doubt whatever that most great men have suffered much from inward need, and that this mental need or mental distress was one of the chief motives of their activities. Nay more, the impetuous and unresting impulse to activity which is characteristic of many persons predisposed to mania may be the motive force leading to great achievements. But if such an impulse to activity leads, as it often does, to friction, we must regard it as morbid.

A morbidly intensified self-consciousness, such as exists in many persons of manic, hysterical, and paranoid temperament, may embolden its possessor to undertake enterprises which a more modest person would not venture to attempt. Colossal self-confidence is usual in great men; and in many of them, as it was, for instance, in Schopenhauer, Wagner, and Nietzsche, this self-confidence is unquestionably magnified to a morbid degree. But the example of some of our own contemporaries will suffice to show us that excessive self-confidence is not alone sufficient to make people fit for great achievements.

Anyhow, it is altogether wrong-headed to suppose that no sound advance in human knowledge and no beautiful works of art can issue from a morbid mind. It is often far too difficult to decide what is objectively true and what is objectively false, for us to be entitled to set up this distinction as a boundary line between the healthy and the morbid. Erroneous convictions may sometimes be life-giving, may promote self-preservation or species-preservation. Consequently, that a thought is erroneous, does not show that the thinker is diseased; nor does that a thought is correct show that the thinker is healthy. Julius Robert Mayer, for example, proved right, in the end, in the idea (the conservation of energy) on behalf of which he contended so fanatically. In like manner, the discovery of Gregor Mendel, which is of enormously greater practical importance than that of Copernicus, is nowise diminished in its greatness by the fact that it was so laboriously achieved by a man whose mind was out of tune. On the other hand, Goethe's theory of colour, which he himself considered to be the main achievement of his life and which he defended with morbid stubbornness, is untenable.

The interconnexion between the respective endowments of the psychopath and the genius, which we have hitherto discussed only in relation to isolated individuals, can of course be traced in the family history of celebrated persons. Galton had already drawn attention to the "distressingly close relationship between these two phenomena", declaring that he had been astonished to find how common mental disorders were in the near relatives of distinguished men.

Not infrequently the morbid taint would seem to be to some extent held in check in an individual of outstanding talent owing to some particularly favourable constitution of the mind, whereas it displays itself far more strongly in some of this distinguished person's relatives. The Goethe family offers an instructive example.[1] Goethe's father was unmistakably psychopathic; in the poet's sister and in his son, psychopathy developed into actual insanity; and both his grandsons were markedly psychopathic. I think there can be very little doubt that the psychopathic taint inherited by Goethe from his father had a good deal to do with rendering effective the mental gift he had inherited from his mother.

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[1] J. P. Möbius, Goethe, third edition, Barth, Leipzig, 1909.

Most people find it a very distressing idea that the striking achievements and great deeds of those men whom they have been accustomed to honour as heroes of the spirit should be in part the outcome of a morbid taint. The scientist cannot, however, on this account attempt to blink the fact. If we read the life histories of a number of celebrated men from the outlook of the trained medical observer, the impression of a high degree of morbidity is irresistible—and this although in most biographies such details are to a great extent shuffled out of sight or presented in a rosy light. Nietzsche, one of the greatest writers and thinkers and at the same time one of the most relentless self-critics of all ages, writes: "What a torment these great artists are, and the so-called higher men in general, to him who has once found them out!"[1] Must this always be so? Is there no help?

Certainly the racial hygienist will be inspired with caution by a study of such facts. A. Grotjahn writes:[2] "But in the case of the persons whose psychical endowments are anomalous, we must never forget that there are numbered among them, not only the antisocial psychopaths, but also those persons whom we prize for their talent or for their genius and to whom we are indebted for every advance in civilisation." He goes on to infer that "we must put up with the existence of numerous psychopaths because from them, or from the families to which they belong, have sprung the valuable sustainers of civilisation". The reader will remember that at an earlier stage of this investigation we learned that the ultimate criterion of the morbid is concerned, not so much with the preservation of the individual, as with the preservation of the race. Is it not possible, then, that the creative activities of the genius may foster the life of the race even when they (or some of the qualities associated with them) run counter to the chances of individual self-preservation? Such a hereditary equipment

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[1] Jenseits von Gute und Böse, 269.

[2] Soziale Pathologie, third edition, Springer, Berlin, 1923.

(though detrimental to its possessor) would be in the highest sense of the word life-preserving, and therefore fundamentally healthy. It is not necessary that all human beings should be average specimens of their kind. A population consisting exclusively of geniuses would certainly be little fitted to survive; but isolated geniuses may tend very greatly to favour the life of the race. It is, in fact, of positively vital importance to a race that it should from time to time produce persons who will open for it new paths in life.

No doubt there promptly intrudes the awkward question whether those men who are usually accounted among our greatest geniuses (Goethe, for instance) have really by their activities furthered the life of the race. I will not contest the possibility that they may have done so, but I must insist that the contrary is likewise possible, and I therefore shall not venture upon a definite answer to a question which many of our contemporaries are inclined to formulate rather crudely. Still, the problem has to be faced. We cannot evade this question of values. Our business as scientists, however, is not to allow any opinion we may hold regarding values to influence our scientific judgment. We value mental greatness and we also value sound health; and we hope that a time will come when the two may be united in the service of the race.

In concluding this section, I should like to express my conviction that morbidity is not a necessary constituent of genius, but merely one of the accompaniments of the conditions that have hitherto cooperated to give rise to genius. Disease, a condition of reduced vital efficiency, is not essential to genius, and cannot possibly contribute as such to the achievements of genius. The connexion between disease and genius is, rather, indirect. Hitherto in human history, genius has not contributed, or at any rate at the present time it does not contribute, to the self-preservation of its possessor. Consequently, it could not be bred as one of the characters of the species, and has only been able to appear as an extensive mutation (see pp. 490–1) of the kind which is as a rule morbid. There is, however, nothing to conflict with the assumption that genius might also be originated by the accumulation of a number of lesser mutations—that is to say, originated in the same way as that which gives rise to the typical characters of a species. As soon as exceptional talent or genius tended to promote the preservation and the increase of the family stock in which they appeared, they would in course of time bring about a permanent rise in the mental endowments of the population, and ultimately the average would stand at a level which we to-day distinguish as genius. In such geniuses as have hitherto existed, there have been many healthy factors of talent; but such factors have hitherto had to have their energies released by separate loss-mutations, which involved the suppression of normal inhibitions. But this should not necessarily be the case. It is easy to understand that the release of the activities that rank as manifestations of genius may most readily be brought about by the suppression of inhibitions, but the manifestations thus produced can never be perfectly harmonious. By deliberately controlled racial hygiene it will, however, be possible, through the aggregation of perfectly healthy hereditary factors of talent, to produce a race in which the average individual will certainly not be inferior in gifts to those whom we now class as geniuses, and in respect of bodily and mental health will be indubitably superior to these. Such a synthesis of high mental endowment and good health is the aim of racial hygiene.

CHAPTER FIFTEEN

PSYCHOLOGICAL DIFFERENCES BETWEEN THE LEADING RACES OF MANKIND

In Section Two of the present work we showed that the various heredity factors which find expression in the human bodily form are met with in varying frequency in different parts of the world—so that for instance light-coloured eyes, which in northern Europe are found in the great majority of the population, become rarer and rarer as we move southwards and eastwards, until in Central Africa and in the Far East they are not met with at all. In like manner, the heredity factors upon which the constitution of the mind depends are variously distributed in different countries. Factors which thus vary in distribution are known as racial factors, and factors of this kind which are concerned in the production of the mental constitution are not essentially different from other heredity factors in general or from morbific heredity factors in particular. Morbific heredity factors are unfavourable to self-preservation or racial preservation, whereas the normal racial heredity factors favour racial preservation in the conditions in which the race normally lives, and are therefore factors preserved and fostered by selection. The differences between the various racial factors are in general less extensive than those between normal and morbid factors; thus, for example, the mental differences between a Chinese and a Negro are less extensive than those between a normal Chinese and a feeble-minded Chinese. On the other hand, a European born with the mental equipment of an Australian Blackfellow would not be able to make good in the competition of modern life, and would presumably be regarded as feeble-minded. In like manner a Negro born with the slender pigmentary equipment of a Nordic would, in his tropical homeland, have insufficient power of resistance to the sun's rays, and would be rightly regarded as albinotic, that is to say morbid. The members of each race are, as a whole, adapted only to the environment in which that race has been living for a long period of time, and these differences in adaptation have certain relationships to the difference between healthy and diseased, which is also concerned with this matter of adaptation.

A morbid race[1] or stock usually differs from the normal race out of which it proceeds by no more than a few though markedly divergent genes. On the other hand, the main races of mankind are distinguished from one another by many genes, each of which, however, may differ to a very small extent only from the corresponding gene in the other races. Since these racial differences are very numerous though small, the differences between the main races are no less momentous than the differences between morbid stocks and the tribal racial forms from which they have originated. An albinotic Negro, though his skin is white, has the overwhelming majority of his heredity factors identical with those of Negroes in general, and is therefore rightly accounted a Negro and not a white. For practical purposes, therefore, the significance of the morbid stocks or races is not very great, since they never form more than small minorities of a population. What are spoken of as "normal" racial distinctions, however, extend to entire populations.

This is a sufficient answer to F. Hertz's rhetorical question: "Is there, then, a fundamental difference between, let us say, white horses and black ones?" White horses and black horses are only distinguished from one another by one gene, but the white and the black races of the human species are distinguished from one another by numerous genes, many of which unquestionably determine mental differences. Hertz's aim is to show that mental racial differences do not really exist, but he does not succeed in establishing his contention.[2] There is no ground for the assumption that the mental differences between the races of man are less extensive than the bodily differences, and in the sphere of practice the mental differences are of enormously greater importance.

If the only differences between the races of man were bodily, racial problems would be of very little moment; and it is precisely because the mental differences are so momentous that some people incline to exaggerate them and others incline to deny their existence. From the outset there could be no doubt whatever that mental racial differences exist. In each race we find that there are average values in the structure of every organ, the brain included; and therefore for each race there must be an average kind of mental equipment. The question, consequently, is, not whether mental racial differences exist, but of what kind they are and how extensive. The anthropologist, if he wishes to have a real "knowledge of human nature", must on no account shirk this problem.

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[1] A good many years ago Billroth, the famous Viennese surgeon, used to speak of "pathological races". Cf. J. Bartel, Morbidität und Mortalität, Leipzig and Vienna, 1911.

[2] F. Hertz, Rasse und Kultur, third edition, Kröner, Leipzig, 1925.

The substratum of heredity is that the organs tend to retain their peculiarities from generation to generation. Heredity factors are racial factors; this applies to mental heredity factors as well as to physical ones. A very large proportion of the mental differences considered above must, therefore, depend upon racial differences in the narrower anthropological sense of the term. Galton recognised that there was no essential distinction here between bodily and mental differences, and in *Hereditary Genius* he wrote: "The natural ability of which this book mainly treats is such as a modern European possesses in a much greater average share than the men of the lower races."[1] The particular racial abilities or aptitudes are preserved in a mixed population; and just as when we find a number of hereditary bodily peculiarities common to various individuals we presume a common descent, so do we infer a common descent when we find that a number of individuals have like mental aptitudes. We are thus led to enquire concerning the mental peculiarities of those primary races out of whose mingling the mixed populations of the modern world have proceeded.

Of course we can only deal here with average values. The primary races of man were never composed of specimens all exactly alike, but were themselves made up of a great number of stocks, differing a little each from the others. In every race, too, we encounter from time to time persons exhibiting such marked hereditary variations as to distinguish them more sharply from the average members of the race to which they belong than these average members are themselves distinguished from the average members of any other race; this applies, for instance, to such mutations as those which cause feeble-mindedness or other hereditary morbid conditions. As a rule, however, such marked deviations are speedily eliminated by natural selection, and even when they persist for a considerable time the average differences between the races endure.

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[1] Preface to 1892 edition.

The mental differences between the primary races which have formed the constituents of our mixed population can no longer be ascertained by direct examination. We have to content ourselves with deducing the mental characteristics of the primary races from a study of the mental differences existing in populations in which a mingling of bodily racial factors is discernible.

It is dangerous, however, to draw inferences concerning the primary racial aptitudes from the characteristics of peculiarly "typical" representatives, for the selection of those regarded as "typical" already presupposes the existence of certain notions in the selector's mind concerning the mental characteristics of the race. No doubt it is true that the various races are much more clearly distinguished one from another in respect of their highly talented members than in respect of those less gifted. Idiots are found in all races. The lowest level of mental equipment is determined by the possibilities of self-preservation (or socially assisted preservation); the uppermost level of mental endowment is determined by the racial aptitudes. Unfortunately, when we are dealing with individuals possessed of exceptional talent, we can scarcely ever decide with confidence whether such a person owes his gifts to the race to which his bodily type mainly corresponds, or perhaps to some other racial strain. We must, therefore, in our study of mental racial differences, concentrate our intention upon the differences between average members of the respective races, even though here (owing to the extensive mingling that has taken place) the true racial differences do not secure full expression. In groups, whose blood has been little, if at all mixed, it is far easier to draw trustworthy inferences concerning racial mental aptitudes than it is in the case of our own mixed population. Especially do the cultural achievements of the various groups enable us to draw valuable conclusions.

The methods of racial psychology are necessarily those of natural science, so that the racial psychologist must aim at the formulation of general laws. L. F. Clauss,[1] who contests this, fails to see that generally valid conclusions can be secured in no other way. He imagines that he can make the Nordic mind "visible" by Husserl's "phenomenological method". But when, like Husserl, he directs his gaze on the mind, he can have no certainty that what he is contemplating is precisely the Nordic mind. How, indeed, does he know that there is a Nordic mind at all, or an "Eastern", a "Western", etc., mind? He simply assumes the existence of what he proceeds to investigate, and thus deceives both himself and others concerning his "methods".

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[1] Die nordische Seele, Niemeyer, Halle, 1923; Rasse und Seele, Lehmann, Munich, 1926.

The *Neandertal race*, which was diffused throughout Europe during the primal age of mankind, did not get beyond the civilisation of the earlier Stone Age and had to yield place to more highly civilised races even before the neolithic period began. Among the extant races of man, the primitive group to which the Veddahs and the Australian Blackfellows belong is obviously in mental characteristics closely akin to Neandertal man. The Veddahs and the Blackfellows make their livelihood by wandering through the forests and consuming anything edible they can find. They have not learned either to breed cattle or to till the soil, nor (and this is especially characteristic of them) have they developed the art of storing foodstuffs as a provision for times of scarcity. They do not build durable huts or make themselves clothing out of skins, although some of the Australian Blackfellows live in regions where it is fairly cold at times. They make no weapons or tools of polished stone, and are therefore still living in the civilisation of the earlier Stone Age. Attempts to teach them agriculture and to make them settle down have been a complete failure. The chief cause of the inability of these primitive races to attain a higher degree of civilisation would seem to be their lack of imagination, which has been studied by M. Moszkowski[1] in some of the aboriginal tribes of Sumatra, who resemble the Veddahs. Those who hold a more favourable view of the possibilities of such primitive races should be reminded that these latter have had just as long a time as we ourselves have had in which to develop a higher civilisation.

Extremely instructive is a comparison between these primitive races and the extant animals which are most closely akin to man.

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[1] Die Urstämme Ost-Sumatras, "Korrespondenzblatt für Anthropologie", 1908.

Wolfgang Köhler[1] has made extremely valuable observations upon the mental achievements of chimpanzees. Chimpanzees can actually use tools (sticks, stones), and can even to some extent make tools. For instance, exceptionally clever animals would spontaneously join up two bamboos in order to make a rod which could reach farther. In character as well as in intellectual endowment chimpanzees are very like human beings. Köhler tells us that one of the greatest pleasures of a chimpanzee is to tease others of his kind or to tease other animals. A chimpanzee in a cage would hold out pieces of bread to tempt fowls to approach, and would then suddenly poke at the birds with a stick or a strong piece of wire. The difference between chimpanzees and human beings is to be found, says Köhler, not so much in the intelligence as in the material upon which the intelligence has to work, inasmuch as chimpanzees have many fewer ideas or much less imagination. Their minds have little to work upon beyond the immediate impressions of the senses. That is why they have never been able to make even the first steps towards the development of a civilisation. Among us human beings there are feeble-minded persons whose mental endowments are no higher than those of the anthropoid apes. On the other hand, the most highly gifted of human beings are as far in advance of the average members of the primitive races of man as these are themselves in advance of the anthropoid apes. Of all the extant races of man, these primitives are certainly most closely akin to our simian forefathers.

The *Negroes* have made a great advance upon these primitives in this respect, that they no longer live exclusively from hand to mouth. They are cattle breeders and agriculturists, and have also developed some of the handicrafts, such as that of the blacksmith. We have to remember, however, that most of the Negro races are permeated with Oriental or Mediterranean racial elements, so that it is impossible to decide to what extent their present cultural achievements are of Negro origin. As compared with the European races, the Negroes certainly lack foresight. In general, a Negro is not inclined to work hard in the present in order to provide for wellbeing in a distant future. The Negro is more strongly influenced than Europeans by the immediate impressions of the senses, and is therefore much more strongly attracted by gewgaws. According to the nature of his present experiences, he vacillates between a cheerful indifference and a hopeless depression.

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[1] Intelligenzprüfungen an Menschenaffen, second edition, Springer, Berlin, 1921; English translation, The Mentality of Apes.

E. Fischer writes of the Negro:[1] "He is not particularly intelligent in the proper sense of this term, and above all he is devoid of the power of mental creation, is poor in imagination, so that he has not developed any original art and has no elaborate folk sagas or folk myths. He is, however, clever with his hands, is endowed with considerable technical adroitness, so that he can easily be trained in the manual crafts. Foresight and mental independence are scantily developed. Usually the Negro lives cheerfully and light-heartedly from day to day." G. O. Ferguson describes as characteristic of the Negro: fickleness; lack of foresight and reflectiveness; deficient tenacity, spirit of enterprise, and ambition; together with an inclination to be contented with momentary successes.[2] R. M. Yerkes[3] reports the unanimous opinion of American officers in the great war to have been that the Negro was a cheerful, willing, and naturally obedient soldier, but one lacking in initiative and capacity for leadership, and therefore unfitted for responsible positions. Since Negroes yield more readily to immediate impulses than do whites, Negro soldiers more often commit thefts and more frequently suffer from venereal diseases.

In the intelligence tests applied in the United States Army during the war, the Negroes on the average were far less successful than the whites. The different results of the alpha test, which was applied to all those able to understand English, are shown in the following table:

12,586 Officers	51,620 Whites born in the U.S.A.	4162 Whites born abroad	2850 Negroes from the northern States	1709 Negroes from the southern States
139·2	58·9	46·7	38·6	12·4

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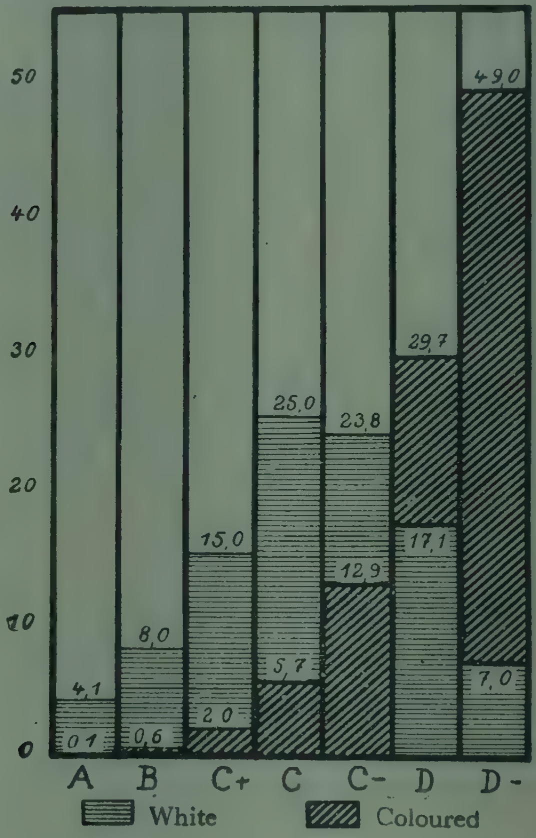
[1] In a contribution to the collective work Anthropologie, Teubner, Leipzig and Berlin, 1923.

[2] The Psychology of the Negro, "Archives of Psychology", New York, 1916.

[3] Psychological Examining in the United States Army, Washington, 1921.

The distribution in the various grades of intelligence is shown in Fig. 171. These grades of intelligence and the alpha marks correspond in the following way:

Talent Class . .	A	B	C+	C	C –	D	D –
Alpha Marks	135·212	105·134	75·104	45·74	25·44	15·24	0·24



A = very good
B = good
C = moderate
D = poor
D – = very poor
C+ = good average
C – = poor average

The figures show the percentage levels attained in the intelligence tests by the white recruits, on the one hand, and the coloured recruits, on the other.

FIG. 171.

Results of the intelligence tests applied to 93,973 white and 18,891 coloured recruits in the American army.

Individuals found unfit for military service because of mental enfeeblement are not included in the records; the proportion of coloured persons rejected on this account was larger. It must not be supposed that the "coloured" are all of pure Negro blood, for a great many of them are mulattoes, etc. Every one with any admixture of Negro blood is counted among the coloured. A number of these "coloured" persons are mainly of European descent, and some of them have a skin as white as that of any European. We must also remember that the Negro ancestors of the present coloured population of the United States, those who were brought from Africa to America, contained a considerable admixture of Oriental and southern European racial elements. On the other hand, in the so-called "whites" there is a certain admixture of primitive racial elements which affects the average of the group. The "whites", too, have now for several generations been subjected to a selection which tends to foster lower-grade endowments, inasmuch as under modern conditions persons with high gifts do not on the average reproduce their kind sufficiently. If we could institute a comparison between pure-bred whites and pure-bred Negroes, the differences would unquestionably be much greater than those shown by the American intelligence tests. Especially does the coloured population of the northern States of the Union contain a large admixture of white blood, for it is the mixed breeds that have tended to migrate from the South into the industrial regions of the North, whereas the comparatively pure-blooded Negroes have tended to remain upon the southern farms. This is the main explanation of the different results of the intelligence tests as applied to coloured recruits in the northern and in the southern States respectively. Since, moreover, most of the Negroes were examined, not by the alpha test (which presupposes a sufficient command of the English tongue), but by the beta test (which does not presuppose such a knowledge), and since the coloured recruits who did not know English well were on the average less gifted than those who did, the difference between white and coloured in respect of intellectual endowment as shown by the test is considerably less extensive than the real difference.

Inasmuch as the objection might obviously be raised that the differences shown to exist by the test might be due to differences in culture as between white and coloured, the material has been subdivided and regrouped in accordance with equality of educational facilities. The results are as follows:

SOLDIERS WHO HAD STUDIED FOUR YEARS IN THE ELEMENTARY SCHOOL

	2773 Whites born in the U.S.A.	355 Whites born abroad	312 "Negroes" from the northern States	356 Negroes from the southern States
Marks ..	23·4	26·6	19·8	8·4

SOLDIERS WHO HAD STUDIED EIGHT YEARS IN THE ELEMENTARY SCHOOL

448 White Officers	14,899 Whites born in the U.S.A.	928 Whites born abroad	555 "Negroes" from the northern States	144 Negroes from the southern States
108·1	64·4	59·4	50·0	28·9

Even now, however, the data, as thus rearranged, do not manifest the whole difference between Negroes and whites, inasmuch as there is a correlation determined by social selection. The whites who do not get beyond the fourth class in the public elementary schools represent a marked selection for deficient intellectual endowment, whereas the Negroes in the southern States who reach this class in the public elementary schools are selected for special intelligence among their kind, seeing that the majority of southern Negroes have very little school education.

In one camp, the coloured recruits were divided into two groups according to the tint of their skins. The members of the lighter group attained on the average 50 marks, those of the darker only 30 marks. Obviously the members of the lighter group owed their greater intelligence to the larger admixture of white blood. Only with caution, however, and with a certain degree of probability, is a comparatively light skin in a coloured person to be regarded as an indication of higher intellectual endowment. A. L. Crane also found in intelligence tests that coloured recruits generally shaped better in proportion to the lightness of the skin.[1]

E. B. Reuter[2] found that among 139 coloured persons who had attained a considerable reputation in the United States, 135 were of mixed blood; and concerning the residue of 4 it may remain open to question whether their ancestors in Africa may not have contained an admixture of Oriental blood. The famous Booker T. Washington was the son of a white father and a coloured mother.

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[1] Race Differences in Inhibition, "Archives of Psychology", 1923, no. 63.

[2] The Mulatto in the United States, Boston, 1918.

Before the abolition of slavery, persons of mixed Negro and white blood were produced in very large quantities in the southern States. F. Hertz[1] quotes the following from Larousse: "The best blood of the South flowed in the veins of Virginian and South Carolinian slaves, and there is said to have been not a plantation in Louisiana on whose cotton fields there were not to be found the half-brothers and half-sisters, the children, or the grandchildren of the owner kept at work by the overseer's whip." Naturally this extensive admixture of white blood has contributed to raise the intellectual level of the coloured population.

Among coloured schoolchildren in the lower forms, Miss Arlitt[2] found an intelligent quotient of 83; in children of Italian origin, 84; and in children of born Americans, 106. By the intelligent quotient is meant the percentage ratio between the intelligence age as determined by intelligence tests, and the real age. Hawis[3] found among 4163 white and 676 coloured schoolchildren, the following percentage distribution of talent (mental defectives not included):

	Good	Medium	Poor
White children	10·5	60·6	11·4
Coloured children	0·1	24·0	38·2

The difference in talent is much less conspicuous during childhood than it becomes later in life, for coloured children are precocious in their development, but their mental advance seems to be arrested at puberty. In the mental life of the adult Negro, childish traits are conspicuous.

We must certainly regard the widely reported cruelty of the Negroes as one of these childish traits. A Negro's sympathies, especially with animals, are very little developed. Here, it would seem, we are concerned, not so much with deliberate as with unconscious cruelty, with such cruelty as we can often observe in our own children, arising from a want of sympathy. We may suppose, likewise, that the notorious lack of sexual control manifested by Negroes is not so much due to any exceptional strength of the sexual impulse as to a general childish lack of the power of restraint.

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[1] Rasse und Kultur, third edition, Kröner, Leipzig, 1925, p. 390.

[2] Quoted by L. Stoddard, The Revolt against Civilisation, New York, 1922.

[3] Quoted by W. Scheidt, Allgemeine Rassenkunde, Lehmann, Munich, 1925.

All authorities are agreed in extolling the oratorical gift and the liveliness of Negroes. Thus E. Schultz-Ewerth writes:[1] "Only one who has been at a religious service attended and managed by coloured folk can form any idea of the way in which these pulpit orators become intoxicated with the exuberance of their own verbosity or can realise the frenzy they arouse in their congregations." Jazz music, which temporarily at least has effected a conquest of the world, is obviously an outcome of the same hereditary endowment. All observers are agreed in declaring that the musical gifts of the Negroes are exceptional.

The organisational and political faculties are very poorly developed in Negroes. They have never produced any kind of social structure worthy to be compared with those found in Europe and Asia. "In Haiti, civilised Negroes left to their own devices, have relapsed into the condition of Bush Negroes, with a revival of fetishism, etc."[2] Similar remarks apply to the Negro republic of Liberia. Where Negroes live in close conjunction with persons of European origin, as in the southern States of the American Union, the former are invariably to be found among the lower strata of the population, engaged in the simpler kinds of occupations. The coloured persons who make their living as brain workers are almost all such as have a considerable admixture of white blood. No pure-blooded Negro has ever achieved works of genius in the European sense of that term.

Average specimens of the *Mongoloid races* greatly excel the average Negro in mental equipment. Among the Mongols of eastern Asia, agriculture and handicraft attained a high level of development several thousand years ago. The culture of the

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[1] Die farbige Gefahr, "Zeitschrift für Völkerpsychologie und Soziologie", 1925, no. 4.

[2] E. Fischer, op. cit., p. 194.

Chinese empire and the existence of China as an organised State for four thousand years, bears unambiguous witness to the high social gifts of the Mongols. The Japanese and Siamese realms were also created by Mongols. On the American continent, three great empires were founded by races akin to the Mongoloids, the Incas in Peru, the Aztecs in Mexico, and the Mayas in Yucatan. The most highly endowed among the Mongoloid races are unquestionably the inhabitants of the Far East. In the Mongol, it is above all the heredity factors tending to promote an aptitude for social life which are well developed; but, on the whole, he has more capacity for imitation than for invention. His memory is stronger than his judgment. He has, therefore, very little interest in the abstract sciences, and inclines to venerate the past. Whereas the Aryans of Hindustan, who have done such wonders in the domains of philosophy and architecture, have almost entirely neglected their own history, the Chinese have millennium after millennium kept careful records of historical and genealogical data. In California and Hawaii, Chinese and Japanese schoolchildren do quite as well in intelligence tests as children of European origin. Chinese students make as rapid an advance in their studies as do American or European students.[1] The Chinese are distinguished for quick apprehension and an excellent memory, but do not excel in imagination, critical faculty, or abstraction. A Chinese, therefore, may attain distinction as a man of learning, but is not likely to become a great investigator or a profound thinker. Chinese culture consists of the memorising of positive data. A Chinese literate has to master the ten thousand or so complicated ideographs of which the Chinese written language is made up. It is true that the mariner's compass, the breeding of silkworms and the spinning and weaving of silks, the making of porcelain, gunpowder, paper, and the printing press were discovered in China; but the Chinese have never spontaneously developed a mechanical technique comparable to that of Europe—and have not developed a correspondingly murderous civilisation!

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[1] Cho Wang, Is the Chinese Heredity inferior? "Journal of Heredity", vol. XIII, 1922, no. 3.

It remains doubtful, moreover, whether Chinese civilisation and the Chinese State were mainly originated by true Mongols. In Northern China there are millions of persons having a slender build, a narrow head, a narrow face, and a narrow prominent nose, whose general type, therefore, suggests a European origin.

Brilliant thinkers, inventors, and investigators—men of genius as we know genius in Europe—can hardly be said to have existed among the Mongols. Despite occasional vicissitudes, the history of Chinese civilisation is one of amazing uniformity, and this uniformity has doubtless in a large measure been the expression of the Mongolian character, which inclines to petrifaction in the traditional. The Mongols, too, have no more bent for metaphysics than they have for science. The doctrines of Confucius and Lao Tse are not concerned with metaphysical matters, but with the practical needs of social life. When, in the writings of noted Chinese philosophers, any reference is made to more general problems, the thought becomes extremely obscure and confused. In the Far East, a number of different religions exist quite happily side by side under conditions of mutual toleration. The history of eastern Asia knows little of wars of religion such as those which have lacerated Europe. It accords, moreover, with the practical sobriety of the Mongols and the small degree to which their imaginations are developed, that they know nothing of romantic love in the European sense. Notwithstanding the general impression we have embodied in the popular usage of the words "Hun" and "Tartar", it is a fact that the Mongoloid races as a whole are less savage and bellicose than the European. Alike in respect of body and of mind, the relation of the Mongol to the European is much like the relation of woman to man: for Mongols are receptive rather than creative; and are at the same time frugal, contented, and patient. Neither on the bodily nor on the mental plane are sexual differences so marked among Mongols as among Europeans. In the Chinese, the masculine courage of attack is less conspicuous than the feminine courage of endurance. Chinese sentiment is far less individualistic than that of Europeans. Individual human lives count for little, even to the individuals themselves. Mongols seem to be less sensitive than we are to bodily suffering.

E. Oberhummer[1] refers to the horrible punishments inflicted by Chinese Law, to the hara-kiri of the Japanese, and to the burnings at the stake by the Red Indians; and he considers a proneness to cruelty characteristic of the Mongoloids.

The disinclination of the Chinese for war is doubtless connected with their comparative inertness and impassivity. For the same reason, the Chinese has no taste for sport. His will to power is small. On the other hand, Oberhummer tells us that he excels men of all other nationalities in business ability. A medical practitioner who has lived for many years among the Chinese writes to tell me that every Chinaman lies, even when he seems unlikely to gain anything by it. Unselfish liberality is alien to the Chinese temperament, and material luxury is scarcely known in the country. On the other hand, culture in the sense of accumulation of great stores of knowledge is held in high repute. The Chinese have a strong feeling for outward dignity, and a considerable amount of that spiritual mandarinism which seems to be more or less characteristic of all the Mongoloid races. The Chinese always wants to save his face, and will invariably mask his feelings beneath an equable smile.

The Mongol's most powerful weapon in competition with persons of other races is his extraordinary frugality, which, in conjunction with his great powers of bodily resistance and the tenacity with which he sets himself to earning his livelihood, enables him to live under conditions so exacting that people of European birth find it impossible to adapt themselves thereto. No other race can compare with the Mongoloid in its capacity for sticking to primitive and monotonous labour. Notwithstanding the unmistakable technical superiority of the Europeans (at any rate in the technique of war) there is much to indicate that the future of the earth belongs to the Mongols, for the Europeans persistently misapply their military technique to mutual destruction, and, influenced by false valuations of life, encourage the dying-out of their best racial elements. The Mongols would seem to be to a great extent safeguarded against both these evils by the nature of their hereditary temperament.

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[1] Völkerpsychologie und Völkerkunde, Vienna, 1923.

The Japanese are obviously to a considerable extent permeated by non-Mongoloid racial elements, derived in the first place from the aboriginal population of Japan (the Ainu), and in the second place from Polynesian stocks. This probably accounts for the fact that the Japanese are much livelier and more warlike than the Chinese, and also have more feeling for the beauties of nature. Although in intellectual endowment they are perhaps slightly inferior to the Chinese, since they have no such overwhelming a respect for the traditional as their continental neighbours they have been able in an astonishingly short time to adopt European technique and European methods of organisation.

The Mongoloid Indians of America are inferior in cultural gifts to the Asiatic Mongols. "In the Indian schools of the United States, ability does not manifest itself above a rather low average", writes Oberhummer. These Amerindians, however, manifest a considerable number of Mongoloid mental traits. For instance, in marked contrast to the Afro-americans, they are reserved and taciturn, and have a strong sense of personal dignity.

The warlike inclinations of the Indians of the north-eastern States (the Sioux, for instance), and also their fondness for sport, may perhaps be attributable to a mingling of Nordic racial elements (the Vikings) in pre-Columbian days—to which the existence of certain bodily traits, such as quite un-Mongolian aquiline noses, likewise points.

It is very difficult to say anything definite about the Mongoloid races of Europe, for in these there has been so much admixture of alien blood. Still, it is clear that European populations containing a large infusion of Mongoloid elements (the Russian, for instance) diverge from the predominantly Nordic peoples in the same direction as that in which the Mongols diverge from the Europeans. The European stocks with a strong infusion of Mongoloid blood have a rather heavy mentality; they cling to the traditional; and among them the individual tends to be absorbed in the community. They are a fruitful soil for mass suggestion and for mandarinism. Superstitions of all kinds thrive among them almost as well as in China; formalism and red tape are as difficult to eradicate. A sense for the beauties of nature and aptitude for the adoption of advanced technical methods are much weaker than in the regions where the Nordic race predominates. The Russians, with their strong infusion of Mongoloid blood, excel rather in suffering and in endurance than in the action that brings freedom. They have a cyclothymic nature (in Kretschmer's sense), with a melancholic fundamental tone. "The Russian folk has no wish to rule; it is by temperament passive, rather gentle, ready to obey—feminine rather than masculine in character."[1] Russian writers are more distinguished for psychological insight (empathy) than for the power of configuration.

In this connexion I may say a few words about the race described by Günther under the name of "Ostisch" (Eastern) which has in recent years become famous if not notorious, but which seems substantially coincident with the "Alpine" race of other ethnologists. Günther describes it as petty, narrow-minded, spiteful, envious, furtive, crafty, cantankerous, sullen, dirty, devoid of creative energy, lacking any sense of higher mental values, persistent day after day in the most unspiritual activities, and so on, and so forth. Of course, the shining qualities of the noble race of the Nordics stand out all the more conspicuously in contrast to this deplorable picture. Just as the youthful Schiller in *The Robbers* contraposed the hero and the miscreant, so does Günther contrapose a noble race and an ignoble one; and it is upon this artistically developed contrast that the powerful impression produced by Günther's book mainly depends.[2] The mass of his readers have formed their opinion, not as the outcome of such facts as he adduces, but emotionally, and under the stress of moral valuations. This is not meant to imply that there is not a kernel of truth in Günther's teaching, but he has systematised unduly and has pushed his theories to an extreme. E. Fischer's description of the mental qualities of the "Alpine" race is much more favourable than Günther's description of the "Ostisch" race:[3] "The intellectual gifts of the Alpine race are, in general, notably inferior to those of the race previously described [the Nordic], but in certain directions they are better developed.

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[1] F. R. Radosawljewitsch, Eugenic Problems of the Slavic Race, in Eugenics in Race and State, Baltimore, 1923.

[2] H. F. K. Günther, Rassenkunde des deutschen Volkes, eleventh edition, Lehmann, Munich, 1927.

[3] In the collective work Anthropologie, Teubner, Leipzig and Berlin, 1923, p. 151.

The Alpines show an inclination to, and a capacity for, persistent and energetic work; they have considerable intelligence and well-developed community feeling. Even though they lack a soaring imagination, their diligence, energy, and skilful use of opportunities bring them successes. Being suggestible, they are ready to adopt and to develop the methods of others; but fundamentally they have marked tenacity." Tendentious writers like Hertz and Wolff have declared that I myself have described the "Alpine", or "Ostisch" race in terms no less unfavourable than those used by Günther. The statement is untrue. In a review of Günther's books I have expressly stated that I regard his judgment as unjust.[1] Besides, I am by no means convinced of the need for distinguishing an "Alpine" or "Ostisch" race. I hold, rather, that the racial elements in central Europe which are commonly spoken of under one or other of these names, are to be regarded as partly of Mongoloid and partly of Near Eastern origin. Among the Tartars, the Finns, and the Lapps, on the one hand, and among the Armenian and other Caucasian peoples, on the other, we find numerous types which are frequently encountered in central Europe as well. This opinion was expressed before me by Luschan, and has recently been underlined by F. Paudler:[2] "Very general is a hesitation to describe by the right name the 'stocky' physique in Europe, so that ethnologists are fond of speaking of an Alpine race, or simply of 'the brachycephalic race'; but this conscious or unconscious endeavour to get along with the assumption that there is only one brachycephalic race, or even to get along without supposing the introduction of Asiatic elements at all, is a defiance of obvious facts and irresistible conclusions." I therefore see no reason for giving any detailed account of the "Ostisch" or "Alpine" race. Nor do I consider that there are any grounds for distinguishing a special "East Baltic" or "Finnish" race. I agree, rather, with Paudler that the peoples described under this head consist of "a very ancient mingling of the Central-Asian-Uraltaic and the Nordic-Indogermanic world". In respect alike of mental and of bodily characteristics, it presents a combination of Nordic and Mongoloid traits.

The *Mediterranean race* shows a certain restlessness, liveliness, and alertness, as contrasted with the comfortable tranquillity of the Mongoloid race. The peculiar mental qualities of the inhabitants of Sicily, Corsica, and North West Africa, as contrasted with the inhabitants of eastern Europe, on the one hand, and those of northern Europe, on the other, are obvious. The Mediterranean has neither the quiet industriousness of the Mongoloid nor yet the enterprising spirit and the energy of the Nordic. He has in a higher degree than the Mongoloid a sense of form, line, and colour, a taste for gesture, and a fondness for the spectacle of lively and variegated movement. The frivolity of the southerner is a heritage from the Mediterranean race, which makes him take life less seriously than does the Nordic. Polite formulas which are not meant to be interpreted literally play a great part in his life—for instance, extravagant offers of gifts, hospitality, etc., which no one is expected to accept. The southern European has less sense of truth and honour than the Nordic. The Mediterranean is almost as passionately devoted to the moment and to the immediate impressions of the senses as the Negro. Although as a rule his mood is childishly gay, he is apt to be unduly crestfallen when circumstances go against him. Hand in hand with this there goes a strong impulse to manifest his feelings in words and gestures. His oratorical faculty is appropriately great, but carries with it the proneness to be intoxicated by words. Thus both by temperament and character, and also in respect of rational endowment, the Mediterranean occupies an intermediate position between the Nordic and the Negro. We find in him the childishness of the Negro, although less developed.

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[1] "Archiv für Rassenbiologie", vol. XVI, no. 1, p. 99.

[2] Die hellfarbigen Rassen, Heidelberg, 1924.

During the war I had several times occasion to note how soldiers from southern Italy, when being subjected to prophylactic vaccinations which were practically painless, would cry loudly: "Oh, mama, mama!" In Nordic, Mongoloid, or Indian soldiers, such a thing would be inconceivable.

A childlike cruelty is likewise characteristic of the Mediterranean race, the popularity of bullfights being a good example of this. Although the average person of Mediterranean race is endowed with a considerable share of natural cunning, in respect of genuine intelligence he falls considerably behind the average members of the other European races. In the application of intelligence tests to recruits in the United States army, the soldiers of Italian and Greek origin shaped badly (see below, p. 653), although we must remember in this connexion that the inhabitants of northern Italy and the inhabitants of Greece are to a considerable extent derived from other races than the Mediterranean. There were practically no Spaniards or North Africans among the recruits. In South America, the Mediterranean race has shown itself decisively superior to the Indian aborigines and to the imported Negroes; it forms there a superior stratum which rules a population composed of Indian and Negroid elements, although in the tropical areas there is a considerable admixture of Indian and Negroid blood in this governing class. No doubt the Mediterraneans contributed important elements to the various great civilisations which have successively flourished and decayed in the Mediterranean region (the ancient Egyptian, the Mycenean, the Etruscan, the Hellenic, the Roman, and the Mohammedan), but they cannot be regarded as the real creators of these civilisations.

It is very difficult to say how much the Mediterraneans may have contributed to the development of the pre-Aryan civilisation of the western Mediterranean lands—the civilisation which has been so vividly described by C. Schuchhardt.[1] Perhaps the Cro-Magnon race gave the main initiative here. Fischer opines that the Mediterranean race may be in some way connected with the rise and spread of the Hamitic languages and civilisations, whereas Paudler says that "as linguistic creations of the Mediterranean race the Semitic tongues alone have to be seriously considered". For the present the question must remain open. In conjunction with other races, and especially with the Nordic race, the Mediterranean race may be a vivifying element in civilisation. For the Nordics, the Mediterraneans embody the essence of the south, for which the Nordics so often yearn. If the Mediterranean is superficial where the Nordic is profound, if the Mediterranean is frivolous where the Nordic is serious, if the Mediterranean lives in the present when the Nordic is anxious about the future—the lighter qualities of the southerner help the northerner to bear the weight of his own. As an example, I may allude to Nietzsche's enthusiasm for Bizet's *Carmen*; but this enthusiasm can, of course, be nothing more than a transient though pleasurable illusion.

In great measure I agree with Günther's description of the Mediterranean race. Clauss, too, has seen far into the Mediterranean mind, but unfortunately this author's writings contain so much that is trifling and unsatisfactory that in general they tend to lower the prestige of racial psychology.

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[1] Alteuropa, Trübner, Berlin, 1919.

The *Oriental race*, which may probably be regarded as a special branch of the Mediterranean race, is distinguished alike for its shrewdness and for its energy and enterprise. It has, however, little fondness for the quiet and persistent toil of agriculture, being inclined rather towards nomadism. It has also produced bold navigators like the Phoenicians. To the Phoenicians is ascribed the invention of the alphabet, marking an enormous advance upon ideographic writing. Furthermore, the so-called Arabic numerals, upon which our modern decimal system of notation depends, would seem to have been discovered by the Oriental race. Oriental racial elements constituted a ruling caste throughout a great part of Africa, until their dominion collapsed in the end of the nineteenth century before the advancing European colonisation of the African continent. The Oriental race must be regarded as having been mainly instrumental in producing the ancient Egyptian civilisation, the Phoenician and Punic civilisation, the ancient Hebrew civilisation, and the Arabian civilisation of the Middle Ages. It played a considerable part, too, in originating the Assyrio-Babylonian civilisation. Very extensive, too, has been its influence upon modern western civilisation, this influence being exercised through the instrumentality of the Jews, into whose racial composition the Oriental stock enters very largely.

The *Near Eastern race* adds to a high degree of sagacity, a peculiar faculty for imaginative insight into the minds of persons of other races—an "empathy" which enables the Near Easterns to guide their own footsteps. The people of predominantly Near Eastern race, such as the Jews, the modern Greeks, and the Armenians, are (as Kant already pointed out) distinguished by a peculiar aptitude for trade and commerce. The Parsees would seem to be similarly endowed.

F. Luschan,[1] who has done exceptionally good service in the study of the Near Eastern race, writes with reference to the "well-known business abilities" of Jews: "But this quality is not peculiar to the Jews, being shared by them with other Orientals and especially with the Greeks and the Armenians. This is plain enough from the fact that all over the East the Jews can rarely if ever gain a footing in towns mainly inhabited by Greeks or Armenians. A popular witticism expresses this with customary exaggeration by saying that one Greek is equal to seven Jews, and one Armenian equal to seven Greeks, which, literally interpreted, would imply that an Armenian is forty-nine times as shrewd a man of business as a Jew." The development of these peculiarities may, perhaps, have been especially favoured by a habitat in Asia Minor, which is the connecting link between Africa, Asia, and Europe. Luschan denies that the Jews have any mental characteristics peculiar to themselves, contending that the "Jewish" psychological traits are common to all the Near Easterns.

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[1] Völker, Rassen, Sprachen, Berlin, 1922.

All the Near Easterns tend to live as minorities in regions mainly populated by other races. The Near Eastern race has been selected to excel, not so much in the control and the exploitation of nature, as in the control and the exploitation of man. Consequently, notwithstanding their high intellectual gifts, the Near Easterns have little capacity for the formation of States. Luschan writes of the Armenians: "Probably there has never been any other people so foolish politically as the Armenian, so incapable of either ruling itself or of being ruled by others." When living in community with other races, however, the Near Eastern is certainly not a mere ferment of decomposition, but becomes a notable stimulant to progress. The earliest of the great civilisations originated in the Near Easterns' domain. In that region, unquestionably, began the use of metals (including that of iron) obtained from mined ores. As to the invention of the alphabet and of Arabic numerals, it is really very hard to decide whether the credit belongs to the Near Eastern or to the Oriental race. The two are close neighbours, and it is not improbable that to a large extent they have a joint hereditary equipment. At any rate the comparatively abstract alphabetic writing and Arabic system of numeration, which are of such overwhelming importance to practical business affairs, are in conformity with the spirit of the Near Eastern race, whereas picture-writing and the Roman system of numeration correspond rather to the visualising and matter-of-fact mentality of the Nordic race.

No doubt the Near Eastern race is inferior to the Nordic in imaginative power, but it is by no means inferior in capacity for abstraction; in formal logic and syllogistic reasoning or dialectics the Near Easterns excel the Nordics, as also in imaginative insight (empathy), alertness, and mobility of mind. On the whole, their mentality is less masculine than that of the Nordics.

Luschan believes that the Near Eastern race created the Alarodic (Caucasian) tongues, and Paudler shares this opinion, which certainly has much to be said in its favour. Beyond question, the Near Eastern race played a predominant part in the development of the Assyrio-Babylonian and ancient Hebrew civilisations, a somewhat smaller part in the development of the Etruscan civilisation and the Hellenic civilisation, but a more decisive part once more in the development of the Hellenistic civilisation.[1] O. Seeck[2] has drawn attention to the fact that almost all the celebrated writers of the Hellenistic period derived from the East. I should like to add that the chief sustainers of the Stoic, the Neoplatonic, the Alexandrian, and the primitive Christian doctrine were of Near Eastern origin. The Hellenistic culture is almost entirely devoid of creative power, thus corresponding to the peculiarities of the Near Eastern race, which is itself interpretative rather than creative. In the modern world, the influence of this race has again become almost as strong as it was in the Hellenistic epoch.

The Near Eastern guides himself rather by the sense of hearing (which tells us more about the mental than about the bodily) than by the sense of vision (which tells us more about the bodily than about the mental). In oratorical faculty and in a capacity for expressing his thoughts and feelings by gestures and play of countenance, the Near Eastern excels persons of all other races. On the other hand, his sense of form is slender. Near Easterns are extremely musical; indeed, I think the Near Eastern is the most musical of all races.

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[1] The Hellenistic civilisation was the civilisation of those who used the Greek language but were not of the Greek race.—TRANSLATORS' NOTE.

[2] Geschichte des Untergangs der antiken Welt, fourth edition, Berlin, 1922.

A remarkably large number of the greatest musicians have many Near Eastern traits. A strikingly large proportion of celebrated musicians have been Jews (Mendelssohn, Meyerbeer, Offenbach, Rubenstein, Joachim, Mahler, Korngold, Auber, Mascagni, Leoncavallo) or half-Jews (Halévy, Bizet). Musical talent is closely connected with the capacity for imaginative insight into the minds of others (empathy).

An inclination to sensuality and cruelty, which is apt to be ascribed to the Near Easterns, is probably connected therewith. Whilst imaginative insight (empathy) is a faculty which enables its possessor to feel another's pain as if it were his own, it may also facilitate the taking of a voluptuously cruel delight in another's pain. Shakespeare depicts an extreme instance of this type in Shylock. On the other hand, the commandment "Thou shalt not kill" is a typical expression of the Near Eastern mind. The Nordic is more simple and direct in this matter.

In philosophy, the Near Eastern inclines to an optimistic monism. It is he who has created the monotheistic religions. In all these religions, the world is represented as being the work of an all-good God. Evil and suffering, no less than good and joy, are used by God for working out his purposes, and therefore must not be taken tragically. Evil is only one manifestation of the divine energy; and, like all the other manifestations of that energy, it will ultimately, as part of God's great plan, lead to good.

I do not consider it necessary to assume the existence of a particular "Dinaric" race. Paudler, too, thinks that what is called the Dinaric race is only a mingling of other races. Günther says: "The Dinaric race would appear, like the Nordic, to be characterised by pugnacity and efficiency, and also by a considerable aptitude for mercantile pursuits. It seems to be unduly excitable, prone to fierce outbursts of temper, and therefore quarrelsome." Again: "The Dinaric has usually a good knowledge of human nature, and excels in apt verbal descriptions of the humorous aspect of our life." Günther also refers to the Near Eastern talent for acting and for music, and says it cannot be regarded as a chance matter that musicians so often exhibit Dinaric traits. These remarks seem to me true enough, but all the traits of which he speaks are such as we know to be partly characteristic of the Nordic, partly of the Near Eastern, and partly of the Mediterranean race—that is to say characteristic of those races whose bodily peculiarities are met with variously combined in the "Dinaric" area. The Bosnian and Slovene pedlars, who are especially good specimens of what is known as the "Dinaric" type, resemble the Near Eastern pedlars in their essential nature as well. Just as the bodily characteristics of the Near Eastern race are not found exclusively in the Balkan peninsula, but spread across Central Europe and even as far as France, so do the mental characters of the same race spread west across Europe. An inclination towards optimism is found also in "Dinaric" Central Europeans. In these regions, moreover, illegitimate children are especially numerous. Among the Southern Slavs and among the French, the sexual occupies the foreground of the mental life quite as conspicuously as among Jews and other Orientals. The "esprit gaulois" is not characteristic of the Nordic Celts, but it is characteristic of the before-mentioned groups. For instance, it seems to me that Rousseau exhibited typical traits of the Near Eastern nature in his ardent erotic fantasy, his amazing power of imaginative insight, and his demagogic influence. Rousseau himself regarded the Nordic temperament as contrasted with his own; and in the Anglo-Saxon husband of his Julie, he attempted to depict the Nordic type, though in doing so he made the mistake of supposing that Nordic reserve and self-command are indications of a lack of deep feeling. The Nordic-masculine was repugnant to him, and his works have in general a somewhat feminine trend, just like the works of Near Eastern writers.

The *Nordic race* created the Indo-Germanic (Aryan) languages and civilisations. Thus the Aryan civilisation of Hindustan unquestionably arose as the outcome of the conquest of the Indian peninsula by the Aryans during the latter half of the second millennium before Christ. During the Vedic period, certainly, these invaders were still mainly of Nordic race. Of course they never formed more in Hindustan than a thin ruling stratum, which held sway over a population racially akin to the Orientals and the Near Easterns. When the Nordic blood became diluted by mingling with the original inhabitants, and when the proportion of Nordic elements was reduced by climatic selection, the creative energy of Indian civilisation progressively declined. In the flourishing and powerful days of the ancient Persian realm, the sustaining force was that of a closely related branch of the Aryans. The doctrines of Zoroaster are a typical creation of the Nordic spirit. Here, in like manner, the decay of the Nordic ruling caste was followed by an atrophy of the civilisation. The blossoming of the ancient Hellenic civilisation came as a sequel to the immigration of the Nordic forefathers of the Hellenes. The statues which have come down to us from those times show clearly that during the first centuries of Hellenic culture the ruling caste was of Nordic race.[1] Furthermore, various writers have carefully described the racial characters of the ancient Hellenes. For instance, Polemon reports that the Hellenes, where their race remained pure, were tall men with fair skins and blond hair. At a later date, Aristotle said much the same thing. The old Hellenic culture, like the ancient Indian and the ancient Persian, declined with the disappearance of the Nordic racial elements. The Roman empire was rooted in the activities of the Italics, a people of the Celtic kinship, and, like the other Celts of that day, of Nordic race.

An amusing confirmation of the statement that the ancient Romans were of Nordic type is to be found in an account given by a journalist named Albert Rapp concerning the statues in the Naples Museum. It appeared in the "Münchener Neueste Nachrichten" of February 26, 1924: "The surprising thing in this collection is the Prussian character of the Roman portrait-busts. The parallelism in the historical situation of the ancient Romans and the modern Prussians has frequently been pointed out, and did actually lead to the perfection of the same type. There are still a few characteristic Englishmen among the Roman busts, but the majority of such busts (thanks to the working of a sound instinct) have long since come to find a place in Anglo-Saxon museums. The busts that I saw in Naples had for the most part soldierly countenances, some of them having a typically Brandenburg look." Rapp, swayed by the current doctrine that the effects of environment are all-important, naturally failed to consider the possibility that what had happened here was, not that a "historical situation" had created a type, but, conversely, that a type had created a "historical situation". His testimony, therefore, is all the more remarkable because he sets out from a standpoint which is the very opposite to our own.

The Teutonic realms which were founded as an outcome of what are termed the folk migrations were founded by Nordic stocks. The German empire of the Middle Ages was entirely sustained by the Teutons. During these same Middle Ages, extensive coastal areas of Europe were ruled by seafaring Normans. In the Italian Renaissance, the blood of the Lombards was a dominant influence. In the soil of the Eastern Empire, which was conquered by the Mongoloid Turks and not by people of Nordic stock, no Renaissance bloomed. Nor did the Iberian peninsula (which in comparison with the two other southern European peninsulas experienced in the early days of its history no more than a moderate amount of Nordic immigration) undergo, in the classical age, any comparable flowering of civilisation to those which took place in ancient Greece and ancient Italy. But towards the close of the Middle Ages, and after the Visigoths had made their way into Spain, there arose that Spanish world-empire "upon which the sun never set". It is plain, in like manner, that the admixture of Nordic blood was of decisive importance in the discoveries and conquests of the Portuguese. The Reformation was born out of the peculiarities of the Nordic spirit, and, by and large, it became established only in countries where the population was predominantly Nordic. The numerically small population of the Netherlands performed wonderful feats in the way of navigation, and founded a large and flourishing colonial empire. Under Gustavus Adolphus and Charles XII the Swedes, also comparatively few in numbers, exhibited a remarkable faculty for political expansion. The French power of past centuries rested on the shoulders of the offspring of Franks, Goths, and Normans. The huge Russian empire was founded by Norsemen (Varangians). The British colonisation of North America, South Africa, and Australia, during the last three centuries, effected by the descendants of Saxons and Normans, represents the mightiest diffusion of the Nordic stock which has occurred since the days of the folk migrations—the mightiest in the annals of mankind. Aryan languages and cultures are to-day spread abroad, not only over almost the whole of Europe, but also over North and South America, South Africa, Australia, India, and considerable parts of Asia Minor and Siberia. The Nordic race, on the other hand, has practically disappeared out of many of these regions and is everywhere on the way to disappear.

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[1] Anton Hekler's book, Die Bildniskunst der Griechen und Römer, gives an abundance of pictorial material, which is all the more convincing because the author was not guided by genetic considerations in the choice of his illustrations.

Modern western civilisation is far from being equably distributed throughout the world, being mainly concentrated in those countries where the Nordic race greatly predominates or in those where there is at least a considerable admixture of Nordic blood. The great scientific discoveries, the most important inventions, and the other mental acquirements of the present day are almost all derived, either from the north western part of Europe (including Finland, Austria, and Switzerland) or from North America.

It is indisputable that the population of southern Europe is less well equipped mentally than that of northern Europe, and the population of eastern Europe than that of western Europe. Compare, for instance, the southern Italians with the Scandinavians, or the Tartars with the Anglo-Saxons. Galton estimated the mental endowments of the population of northern England and of Scotland at one degree higher in his classification than the population of the rest of England, where there is a considerable infusion of Mediterranean blood. He found that the number of celebrated men born in the north of England was far greater than was proportional to the population. A. Odin's enquiries give similar results regarding the origin of the great men of France, a majority of whom have derived from the regions settled by the Nordics.[1] Ripley[2] has shown the facts graphically, in a map. In the United States, J. Mc.K. Cattell[3] and F. A. Woods[4] have found that to a preponderant extent those men who play a leading part in the cultural life of the Union are the offspring of nations which are mainly of Nordic stock, and that, when not thus derived, they are generally of Jewish blood. L. M. Terman,[5] studying the family trees of 1000 talented Californian children, obtained similar results. With very few exceptions these children were either the offspring of Nordics from the north-western half of Europe or else were of Jewish descent. Among these talented children, there were practically no representatives of the southern Europeans or of the coloured races.

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[1] A. Odin, Genèse des grands hommes, Paris, 1895.

[2] W. Z. Ripley, The Races of Europe, second edition, New York and London, 1910.

[3] A Statistical Study of Eminent Men, "Popular Science Monthly", vol. LXII, 1903.

[4] The Racial Origin of successful Americans, "Popular Science Monthly", vol. LXXXIV, 1914.

[5] Genetic Studies of Genius, vol. I, Stanford University Press, 1925.

Geographical distribution apart, we can use religion to assist us in the indirect grasp of racial distinctions. A. de Candolle[1] has found that among the members and corresponding members of the most noted scientific societies, the proportion of Protestants is almost four times as great as might have been expected in view of the proportion of Protestants among the population of Europe. He considered that the differences in religion directly accounted for this, but the connexion is more probably indirect, inasmuch as a larger percentage of Protestants are Nordics than members of any other race. We must, however, also bear in mind, that for many centuries the Catholic populations in every generation have lost a large proportion of their intellectual endowments through the celibacy of the clergy.[2]

The intelligence tests applied to American recruits throw light upon the differences in mental gifts among the European races. The subjoined table (p. 652) shows the distribution of the various degrees of talent among the recruits born in Europe.

The percentage figures are taken from the official report issued by R. M. Yerkes;[3] the "intelligence age" was calculated by Professor Brigham of Princeton University.[4]

When the countries of origin are arranged in the order of the average talent of the recruits examined, we get the picture shown in Fig. 172. The number following the name of each country gives the "intelligence age", the horizontal numbers give the percentages in which the various degrees of intelligence were represented in the different groups. Countries from which less than 100 recruits had been derived, have not been included in the table; that is why, for instance, France and Spain do not appear in it.

We must not, of course, make the mistake of supposing the mental endowment of the recruits to represent without

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[1] Histoire des sciences et des savants depuis deux siècles, Geneva, 1873.

[2] Nor must we forget the effects of the Inquisition in eliminating certain higher intellectual types.—TRANSLATORS' NOTE.

[3] Psychological Examining in the United States Army, Washington, 1921.

[4] R. M. Yerkes, Eugenic Bearing of Measurements of Intelligence in the United States Army, "Eugenics Review", vol. XIV, 1923, no. 4.

	Belgium	Denmark	Germany	England	Greece	Holland	Ireland	Italy	Norway	Austria	Poland	Russia	Scotland	Sweden	Turkey
Number of Cases	129	325	299	411	572	140	658	4007	611	301	382	2340	146	691	423
A	—	0·6	3·3	5·6	—	5·0	1·2	0·2	0·6	1·7	—	0·4	4·8	1·3	0·4
B	0·8	4·8	5·0	14·1	2·1	5·7	2·9	0·6	3·5	1·7	0·5	2·3	8·2	3·0	3·0
C +	11·6	16·2	17·6	24·0	2·1	21·4	8·4	2·3	8·9	6·7	3·1	4·8	25·4	12·6	5·7
C	39·2	32·4	31·8	12·4	36·7	25·0	29·0	24·4	36·0	32·3	19·5	22·1	19·2	37·0	34·4
C –	24·0	33·0	27·8	35·4	15·7	33·7	18·6	9·1	25·9	20·0	7·3	10·5	28·8	26·8	14·7
D	18·6	12·8	11·7	6·0	35·1	8·5	26·2	40·0	21·8	27·5	43·5	40·0	10·9	17·1	30·4
D –, E	5·4	0·6	3·3	2·7	8·5	0·7	13·2	23·4	3·8	10·0	26·4	20·4	2·7	2·3	11·6
Average Intelligence Age	12·8	13·7	13·9	14·9	11·9	14·3	12·3	11·0	13·0	12·3	10·7	11·3	14·3	13·3	12·0

qualification the average mental endowment of persons in the lands from which they are respectively derived. We must suppose that those who have emigrated to the United States have been subjected to a certain selection, which may in some countries have been favourable to quality and in others unfavourable. In the smaller groups, too, the error of small numbers may play a certain part. The premier position occupied in the table by immigrants from England and Scotland may be largely due to a special selection of these immigrants. The practice of the higher occupations in the United States generally presupposes a thorough command of the English language, and the result of this is that persons belonging to the higher classes can only as a rule find appropriate occupations in America when they come from an English-speaking country. Since, moreover, the higher occupations represent per se a selection for higher intelligence, it is easy to understand why

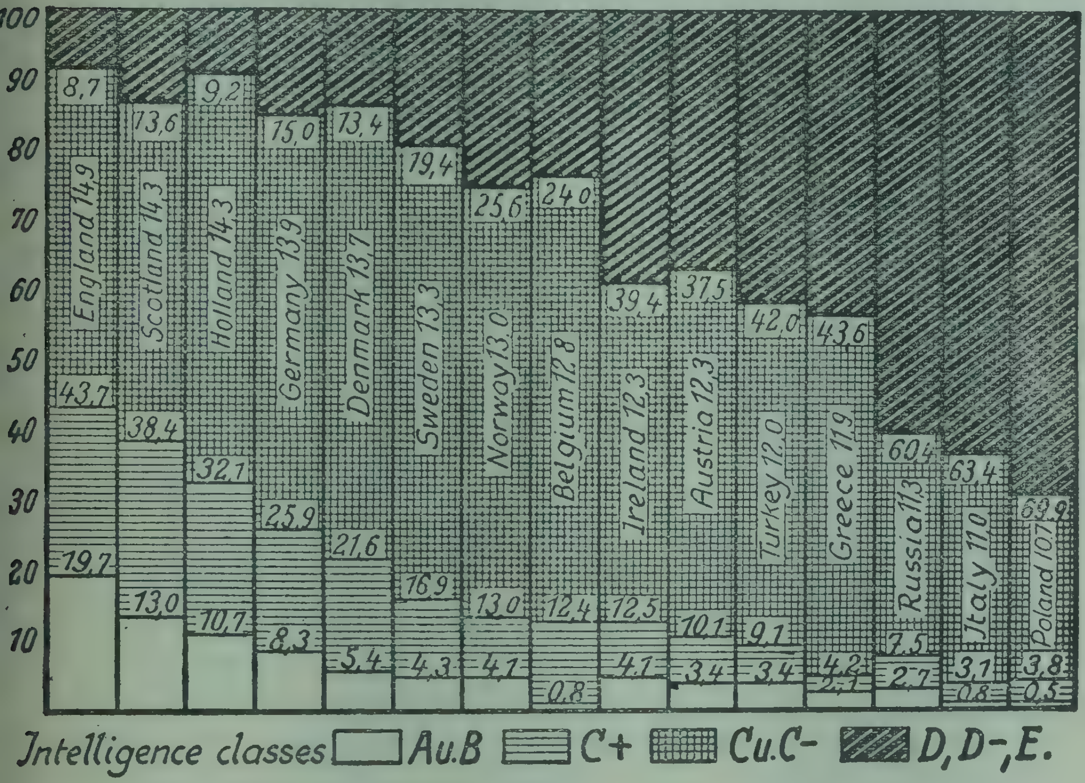

FIG. 172.

Results of the Application of Intelligence Tests to 11,435 United States Army Recruits born in Europe.

there should be a considerably larger proportion of persons well endowed mentally among the immigrants from England and Scotland. Immigrants from other countries, on the other hand, can usually make a livelihood in the United States only as manual workers or in other subordinate positions, or when they settle on the land as farmers. That probably explains why the immigrants from Scandinavia occupy a less favourable place in the figure. In view of these considerations, the position occupied in the figure by the Irish immigrants, most of whom speak good English, must be regarded as a remarkably bad one, and as indicating that the Irish are on the average far less well endowed mentally than the Scandinavians. The poor position occupied by the "Russians" and the "Poles" is all the more striking inasmuch as most of the recruits classed as coming from Russia and from Poland were probably Jews. The fact is, however, that Jewish immigrants to America generally make a livelihood, either as tailors or as practitioners of some similar petty handicraft, or else as small traders. This involves, as regards intelligence, an unfavourable selection of the immigrants. Furthermore it has to be remembered that the intelligence tests mainly concerned the visualising faculty, and therefore do scant justice to the abstract gifts of Jews. Among the "Austrians", too, there were many (Galician) Jews. The term "Austria" is used in its pre-war signification. Probably the German Austrians, separately considered, would have shaped better in the tests. In a comparison between England and Germany, we must remember that Scotland and Ireland were treated separately from England, whereas Germany was treated as a whole. Had the separate countries in Germany been separately treated, some of them would probably have shaped quite as well as England and Scotland. It must not be forgotten that the genuinely Anglo-Saxon elements among the British derived originally from North West Germany.

Making due allowance for these various considerations, we draw the following conclusions. *The countries belonging to the north western half of Europe are without exception in the first half of the series.* But these are the countries containing the largest admixture of Nordic blood; or, to speak with more precision, the largest admixture of the slender blond race and of the heavily-built blond race.

The foregoing data may be supplemented by giving the results of intelligence tests applied by Dickson to Californian schoolchildren.

	Intelligence Quotient
37 children of Spanish descent	78
23 ,, ,, Portuguese descent	84
25 ,, ,, Italian[1] descent	84
14 ,, ,, N.W. European descent	105
49 ,, ,, U.S.[2] descent	106

The intelligence quotient is obtained by dividing the "intelligence age" by the actual age. Since children of the Mediterranean race ripen earlier than children of the Nordic race, we may suppose that adults would manifest differences greater than those shown above.

I do not think it would be an exaggeration to say that in respect of mental gifts the Nordic race marches in the van of mankind. In creative mental power the Nordics would seem, on the average, to excel the Near Easterns and the Orientals, although these latter are as good as the Nordics in power of intellectual grasp. Still, some of the Near Eastern and Oriental elements among the Jews are fully equal to the Nordics in many domains of intellectual life, and are superior to them in some. For the rest, the Nordic race owes its leading position in the world, not only to its high intellectual endowments, but also to its character traits.

E. Fischer writes:[3] "The mental endowments of the Nordic race are great energy and industry, vigorous imagination, and high intelligence. Conjoined with these are foresight, organising ability, and artistic capacity (this being least marked in respect of music); and also the unfavourable qualities of strong individualism, a lack of community sense and of willingness to obey orders, a certain one-sidedness, and an undue inclination towards meditation and flights of fancy, a dislike for steady and quiet work; while as additional qualities may be mentioned, a considerable expansive force, the power of devotion to a plan or an idea, an inadequate capacity for instilling an idea into others, and a small inclination for adopting the ideas of others—in a word, scant powers of suggestion and comparatively little suggestibility. It is obvious that when circumstances are favourable, persons richly endowed with these gifts are likely to become leaders, inventors, artists, thinkers, organisers."

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[1] For the most part from southern Italy.

[2] Mostly of Nordic stock.

[3] Anthropologie, Teubner, Leipzig and Berlin, 1923, p. 150.

Least of all human beings is the Nordic the slave of the moment, for he excels the members of all other races in constancy of will and in foresight. Owing to his Promethean character, he inclines to subordinate his sensual impulses to a remote aim. Self-control is, perhaps, the most distinctive among the characteristics of the Nordic race, and upon this depends in large measure the Nordic gift for civilisation. Races lacking in self-control are unable to be guided by long views and to pursue remoter ends.

These mental peculiarities of the Nordic race are obviously connected with the northern environment—not, however, in the sense that the damp and cold climate of the north has directly fostered the Promethean faculty; but, rather, in the sense that families characterised by the Southland levity, families not well equipped with the faculty of providing for a rainy day, would much more often perish during the long northern winter. Thus race is, to a degree, the product of environment, but is not the direct product of environment in the Lamarckian sense of the term, inasmuch as a selective process is involved.

Of essential importance, therefore, are the primary developmental possibilities of a race. The Mongoloid races, like the Nordic races, have been adapted to the northern climate by selection. But whereas in Nordics the power to cope with the inhospitalities of the northern environment has been obtained by an increase in the intellectual capacities, among the arctic Mongoloids natural selection has picked out those who had the fewest wants.

To selection by the northern environment, the Nordic doubtless owes likewise his gift for technique, and, in general, for the mastery over nature. Persons who wish to keep themselves alive in the northern environment must be able to build durable houses and seaworthy ships. That is why most technicians and inventors to-day belong to the Nordic race. The Nordics are more highly gifted for the natural sciences than for historical and literary studies. I have been struck by the fact that among the students at the technical academies the Nordic type is more prevalent than among the students at the older universities. We see this type especially often, too, among carpenters and architects.

During the Ice Age, the ancestors of the Nordics, inhabiting the ice-free region to the north of the great chain of mountains stretching from the Eastern Caucasus to the Western Pyrenees, lived almost exclusively by the chase. With the primitive weapons of the Stone Age they were able to attack and overpower, not only the gigantic and clumsy mammoth, but also the powerful cave bear. The environmental conditions necessitated courage, combativeness, and contempt for death, and these were the qualities that favoured survival in such primitives. Uebel[1] shows with fine insight how the wandering instinct of the Nordics, their susceptibility to the call of the distant and the unknown, were fostered by the conditions under which the hunters of the Ice Age lived. "The period of the primal migrations was doubtless the birth hour of the Nordic soul."

Characteristic, too, is the fondness of the Nordics for the sea. Quite early in the Middle Ages, Nordic Vikings set forth across the boundless ocean, voyaging by way of Iceland to North America. Apart from the Nordics, only a very few kindred stocks were able to build ships rendering deep-sea voyages possible (the ancient populations of the western shores of the Mediterranean, the Phoenicians, and the Polynesians). For the Negroid and Mongoloid races, and also for the Near Easterns, the sea remained an uncongenial element, whose waters could not be bridged. The ancient Hellenes derived their taste and their gift for seafaring from their Nordic ancestors; from the same source came their sense for the plastic arts and for architecture, and also their gift for profound investigation; but part of the same heritage was their lack of community sense, their deficient capacity for forming themselves into one great union—a defect which became for them a doom.

The Nordic environment was not one which allowed human beings to dwell in great communities. In view of the scarcity of food, only small kinships could outlive the northern winter. The result was that in the Nordic race there was fostered by selection an inclination towards isolation. In that rigorous environment, a tendency to keep away from neighbours, a readiness to pick a quarrel, was actually favourable to preservation. Beasts of prey live alone, or at most in very small communities, and that was the way in which man lived during the Ice Age. But the qualities which promoted self-preservation and species preservation then, no longer do so now.

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[1] Nacheiszeit und Nordische Rasse, "Archiv für Rassenbiologie", vol. XVIII, 1926, no. 4.

People are fond of contrasting the Nordics as settled agriculturists with the wandering peoples which have persisted in a nomadic life. Really, however, the Nordic is less of an agriculturist than is the Mongoloid. The Vikings must be regarded, rather, as the typical Nordics. No one can deny that the mental endowments of the Nordic have promoted his activities as a pirate. The famous Icelandic sagas are full of violence and murder. Nevertheless the Nordic is not, properly speaking, cruel. He does not take positive delight in others' suffering, and his cruelties are merely the expression of the combativeness and courage of one who sets his own life, too, at a low valuation.

The Nordics are not inclined towards the "ressentiment" which Nietzsche regarded as characteristic of lower races. No doubt the Nordic can hate, but as a conqueror he is conciliatory, and so is the true Anglo-Saxon. The *Song of Hate* against England which had considerable vogue in Germany during the war was not composed by a Teuton, for its author, Lissauer, was a Jew.

Unfortunately, the Nordics are inclined to regard war as a sport and to practise it as such. In the description of the battle of the Skagerack, the main interest turns upon who remained in possession of the battlefield, although really this did not matter a jot, seeing that the "victors", too, would have to go home again as soon as the hurly-burly was over. Happily, however, we need not suppose that pugnacity is an ineffaceable characteristic of the Nordic, for it may be hoped that his powers of insight (which are likewise exceptional) will in the end convince him that persistent self-mutilation is senseless. The growth of such an insight probably explains why the modern Scandinavians are no longer pugnacious. The inclination for sport is closely akin to the fondness for making war. Nordics constitute a disproportionally large number of sportsmen. To some extent it would seem that danger as such is attractive to the Nordic mind, inducing the Nordics to devote themselves to mountaineering, skiing, and similar risky sports. The Nordic fondness for the sea is still manifested to-day in the taste for yachting. In the sportive and militarist behaviour of the Nordics, their restrained energy (that is to say their capacity for achievement) finds expression.

Nordic courage gives power, not only for warlike activities, but also for great deeds in the mental sphere. The courage to speak truth and ensue it is often of decisive importance to the attainment of knowledge which will prove of far-reaching significance, That is why the Nordic race can boast so proud a list of great thinkers and investigators. The Nordic race has also produced conspicuously great statesmen. Günther reminds us that Treitschke spoke of Lower Saxony as the "land of statesmen". It is a fact that in this respect no other Germanic land can vie with England so successfully as can the original home of the Anglo-Saxons. In addition to his exceptional faculty of judgment, it is, above all, the Nordic's will to power and his fondness for shaping things which endow him with his talent for organisation.

> With typical exaggeration and in his deliberately mortifying way, Nietzsche says that the State originated through the activities of "a herd of blond beasts of prey, a race of conquerors and masters, which, with all its warlike organisation and all its organising power, pounces with its terrible claws on a population in numbers possibly tremendously superior but as yet formless, as yet nomad. Such is the origin of the 'State'. . . . The work of these blond beasts is instinctive creation of form, an imprinting of form; here we have the most involuntary, the most unconscious artists in the world".[1]

The Nordic has little aptitude for exercising a mental influence over other human beings. This is an art of which the Near Eastern is a past master. The Nordic finds it very difficult to put himself in another's place. His instincts are individualistic rather than social, his craving for independence makes him resist being enrolled in a community. The talents and the character of the Nordic are, indeed, of great value in social life; and yet society means very little to him, and he will never merge himself in it.

A certain remoteness from men and things is, as Clauss in especial has pointed out, characteristic of the Nordic. This is what Nietzsche has spoken of as the "pathos of distance". Connected therewith is the Nordic objectivity, for without the power of standing apart from men and things to contemplate them, an objective judgment concerning them is scarcely possible. The Nordic is little inclined to show his feelings; he does not wear his heart upon his sleeve. An aristocratic power of self-restraint saves him and his kind from importunacy. He keeps to himself his thoughts about the problems

[1] Zur Genealogie der Moral, section 17.

that move him most profoundly. Even in matters of love, the Nordic seems always to retain a certain aloofness of mind.

Because the Nordics are self-controlled and reserved, they are often misunderstood, and are believed to be unfeeling; and this opinion of them is diligently circulated by those who do not like them. Such a view, however, is fundamentally unsound. The Nordic is fastidious in his love, but never cold. Frenssen, the Norse poet, who is well acquainted with the Norse mind, lays frequent stress upon the vigour of the sexual temperament among the Nordics, and is right in so doing. The Nordic has little taste for superficial sociability and superficial cheerfulness, but the current of his feeling runs strong and deep. In warm cordiality the Nordic race is inferior to none.

What is known as personality or individuality has undergone its fullest development among the Nordics. Nietzsche once spoke of man as "an animal which can promise something". This applies especially to the Nordic. In the Nordic lands one can trust the word of a manual worker; the trains run according to schedule; the public officials are helpful. In houses of entertainment one can leave one's overcoat in the entry without fear that it will be stolen. Such honesty and straightforwardness are, to say the least of it, by no means the rule in southern lands. Yet the countries where the Nordics predominate have not been favoured by nature, and if people there were dirty in their ways and backward in their civilisation, those who make so much of the influence of environment would not be slow to find the cause in the parsimony of nature. As regards the average level of civilisation attained by its inhabitants, Sweden actually stands in advance of all other countries. Simply as a matter of self-respect, the Nordic wants "fair play" in all competitions; but he is likewise animated by a love of order and by cleanliness of mind. Bodily cleanliness, too, is rightly regarded by Günther as a characteristic of the Nordic races. This is obvious to any one who compares towns and persons in the north and the south. Lice, which are still very common in eastern and southern Europe, are very rare now in the north and the north west, so that during the great war one of the chief miseries of the soldiers of Nordic race was that caused by body lice, which infested the trenches.

The Nordic has more circumstantial interest and less psychological interest than the Mongoloid and especially than the Near Eastern. Extremely marked is his feeling for nature, whereas the interest of the Near Eastern turns especially towards the social life of mankind. The Nordic interest turns rather outwards towards the phenomenal world than inwards towards the mind. The Nordic thinks in visual images; he is, as Goethe writes, "born to see, adapted for vision". He finds beauty, above all, in form. Consequently his artistic gifts find expression chiefly in the domain of the sculptural delineation of form. In marked contrast with the portrait busts of the ancient Egyptians, the classical plastique of the ancient Hellenes (which in the north-west of Europe is down to the present day regarded as the climax of art) neglected the expression of the mind. Not until the later days when the influence of the Near Eastern race became predominant, did Hellenic art devote itself to the expression of the mind.

For music, which expresses the emotions of the soul, I do not consider the Nordic race to be especially gifted. Although it is true that a great many among the most distinguished composers are mainly of Nordic blood, it seems to me that what they owe to their Nordic heritage is rather their mental creative faculty than their musical endowment proper.

As a thinker, the Nordic aims, before all, at clarity. The classical composure and sobriety of the ancient Hellenic and the modern Anglo-Saxon thinkers are genuinely Nordic.

Günther tells us that by his eastern (East Baltic) neighbours, the Nordic is regarded as a cold, arid, unfeeling calculator. The Anglo-Saxons produce the same impression upon the Germans. The desire to achieve a purely rational contemplation of the universe, which we Germans incline (with a somewhat contemptuous implication) to speak of as "rationalism" and which we regard as typical of a civilisation more western than our own is, in reality, an expression of the Nordic spirit—whereas that cannot be said of "German idealism" which delights in magian words and obscure phrases. Genuine idealism is perfectly compatible, nay, almost identical, with the will to rationality. It must not be forgotten that the word "idea" originally denoted a visual image. Nordic idealism is, then, compatible with a healthy realism, that is to say with a sense for the actual and the essential.

"The Nordic", says Nietzsche, "has no inclination for the indiscriminate gratification of the impulse to knowledge"; rather does he demand from knowledge that it shall be related to the essential aims of human reason (Kant). Consequently he is not a polymath. Oberhummer draws attention to the fact that among the Aryans of Hindustan there prevailed "a complete lack of a sense for positive knowledge", whereas the Chinese have a strong taste for this, and have produced an abundance of historical and geographical treatises. A certain deficiency in historical and geographical culture, of which we Germans are apt to accuse the British and the Americans, would seem to be grounded upon the nature of the Nordic race. Nordics have more sense for fundamental generalities and for things which happen in accordance with law than for particulars and for chance occurrences. They therefore produce more natural scientists and philosophers than historians and men of literary learning, more investigators and thinkers than pundits and polyhistors. The Nordic trusts more to his own judgment than to positive knowledge; the Mongoloid, on the other hand, is inclined to prove all things, and favours a comparatively passive acceptance of their peculiarities and individualities; his realism takes the form of a patient entering into the endless multiformity of actual things.

The inclination of the Nordic to get to the bottom of everything; his dissatisfaction with the obvious, his longing to bore into the depths, inclines him to metaphysical speculation. The Nordic yearning is always vainly seeking satisfaction. Connected therewith is the Nordic's peculiar spiritualisation of love, which is apt to have a morbid side, and may endanger the life of the race. The Nordic is prone to a tragical outlook upon the universe and upon human life.

The essential religiousness of the Nordic mind has found its most characteristic expression in the ancient Persian doctrine of Zoroaster. Hormuzd, the god of light and goodness, is at perpetual warfare with Ahriman, the god of darkness and evil, and it is the duty of the good man to be one of the warriors in the army of light. The same mood underlies the ancient Teutonic view that after death the tried and trusted heroes will, in Valhalla, engage in divine combat one with another, and will in due time be called to play their part in Woden's army in the last great fight against the powers of darkness. Life is looked upon as an unceasing task; reality is not an inalterable datum to which we have to accommodate ourselves, but something tainted with inadequacy and something which has to be overcome. That is the essential mood of the teaching of Plato, of Kant, and of Nietzsche. Among the Christian religions, Protestantism is more in conformity with the Nordic spirit than is Roman Catholicism. The Nordic's strong sense of independence, his desire for an individual judgment and for personal responsibility, rise up in revolt against the authoritarian standpoint of Catholicism; and, furthermore, the Catholic religious service, with its appeal to the senses and the emotions, is less congenial to the rationalist spirit of the Nordic than is the comparative sobriety of the Protestant religious service. Of course we are concerned here, not with radical contrasts, but only with differences of degree. Still, Protestantism has never made much headway except in the countries which are predominantly Nordic, and with the decline in the Nordic race we may expect these countries to become Catholic once more.

We must now consider the mental peculiarities of that race of north-western Europe which is distinguished from the slender (leptosome) Nordic race by its ponderous (athletic) build, by a shorter and wider face, a broad forehead, widely separated eyes, and by a lower jaw which is more perpendicular in front and whose angles are closer to a right angle. This type is supposed, rightly I think, to derive from the Cro-Magnon race. Fischer has pointed out that Cro-Magnon types are still to be found abundantly in Lower Saxony (see above, pp. 190–1). That explains why it is that in many parts of North Germany the vertical extent of the face is less than in South Germany —a fact which has hitherto puzzled ethnologists. Since my attention was first drawn to this type, it has become more and more plain to me that in North Germany, in Holland, and in Denmark it constitutes a noteworthy proportion of the population—and in certain areas, as in Westphalia and Frisia, is the main ingredient.

It is no longer desirable to speak of this heavily-built and blond stock as the Cro-Magnon race. It shows no more than a certain likeness to the Cro-Magnon remains in southern France. I think we need a special name for the heavily-built blond race, and I propose to speak of it as the *Atlantic race*, since this type is chiefly found in countries near the Atlantic coast. The fact that Fischer has found kindred forms in the Canary Islands, and the fact that they seem also to occur among the populations of north-western Africa and south-western Europe, likewise speak in favour of the use of this appellation. The French anthropologist Deniker described in the coastal countries of south-western Europe persons of a large, dark type, and spoke of them as constituting an Atlanto-Mediterranean race. Since the name "Atlantic race" did not occur to me until I was revising the present chapter, after Fischer's contribution to our book was already finished, and since I do not know whether the name will catch on, I shall provisionally be content, for the most part, to speak of the "heavily-built blond race".

In the later Stone Age, the creators and sustainers of the megalithic civilisation of north-western Europe obviously belonged for the most part to the heavily-built blond race; but to the same race belonged, in an earlier time, those coast-dwellers who have left us their kitchen-middens as evidence of their settlement for thousands of years upon the coasts of north-western Europe and to some extent also farther south—these kitchen-middens are composed chiefly of mussel-shells. The slender blond race, on the other hand, would seem in the later Stone Age to have created the "band-ceramic civilisation",[1] which, as Paudler admirably shows, spread from a focus lying to the north-west of the Black Sea (the so-called Tripolje civilisation), along the Rhine towards the west, along the Oder and the Vistula towards the north-west, and along the western shore of the Black Sea towards the south-west. Thus the "Nordic civilisation", in the sense of the Stone Age civilisation of Scandinavia, was not originated by the slender blond race, but by the heavily-built blond race in conjunction with the megalithic civilisation. From this it appears that the Teutons must represent a mingling of the heavily-built short-faced primitive population of north-western Europe with the slender long-faced race migrating from the Black Sea region. According to Paudler, the heavily-built blond "pre-Teutons", prior to their Aryanisation by the slender blond conquerors, must have spoken a language belonging to the Hamitic group of tongues. M. W. Hauschild, likewise, has come to the conclusion that the Teutons have been derived from a mingling of these two races.[2]

Since the Aryanisation of northern Europe presumably did not begin until soon after 1000 B.C. (Paudler, p. 79), we need not be astonished to find that in Teutonic tradition there are memories of the days when these two blond races were distinct. In the Scandinavian mountains, where we may suppose remnants of the original inhabitants to have taken refuge from the conquering Indo-Germanic invaders, there is still to be found the "Jötunfjeld", the land of the giants. In North Germany, the barrows, the burial mounds of megalithic times, are spoken of as the "giants' tombs". In Teutonic mythology, the inhabitants of the Middle Realm dwell between the giants of the north and the dwarfs of the south. The cumbrous frost giants, equipped only with weapons of wood and stone, were outmatched by the skilful Thor, the god of agriculture. In the south, on the other hand, there dwelt the cunning black-haired dwarfs, skilled in the arts of extracting ores from the earth and of forging iron. If the original home of the slender blond race was the Black Sea region, we shall naturally be inclined to identify the frost giants with the heavily-built blond race, and the dwarfs with the Near Easterns.

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[1] So called because the pottery they made is adorned by band designs, instead of by mere lines, as in more primitive types of pottery.—TRANSLATORS' NOTE.

[2] Die menschlichen Skelettfunde des Gräberfeldes von Anderten bei Hannover, "Zeitschrift für Morphologie und Anthropologie", vol. XXV, 1925, no. 2.

In our own time, populations in which the heavily-built blond race predominates are distinguished psychologically from those in which the slender blond race predominates. For instance, in certain parts of Lower Saxony and in Westphalia there is unmistakably a certain mental cumbrousness of the population. The heavily-built blond human being is less mobile than the slender type; he lacks the impulse towards the remote; he clings to home and to the traditional. On the other hand, he excels the Nordic in trustworthiness. What we speak of as "German fidelity" is especially characteristic of him. Perhaps it was no chance matter that the Reformation failed to establish itself in Westphalia. The heavily-built blond race is inclined towards musing and mysticism. It is mainly responsible for the German earnestness and depth of character.

The heavily-built blond human being finds it even more difficult than does the slender blond to see into the mind of others. He has even less "empathy". He sticks obstinately to his own ideas. The Westphalians and the Swabians, who apparently contain a strong admixture of the heavily-built blond ingredient, are proverbially stubborn. The heavily-built blond outdoes the slender blond in reserve and taciturnity. It is to this trait that must doubtless be referred the

Teutonic fondness for settling down in isolation on the land. The heavily-built blond wants to be master on his own farm, and prefers to live out of sight of his neighbours. It was on these lines that the Boers colonised South Africa. Hauschild believes that the remarkable impetus of the Teutons must be mainly due to the heavily-built blond racial element; but the heavily-built blonds are less fitted than the slender blonds for leadership and rule. Still, several of the greatest leaders among the Germans—for instance Bismarck and Hindenburg—had many of the characteristics of the big blonds. When the Atlantic ponderousness is coupled with the Nordic boldness, we get figures of megalithic proportions.

The heavily-built blond, like the slender blond, has been selected rather for his visual than for his auditory aptitudes. He, too, is more inclined to be an architect or a sculptor than to be an orator or a musician. The Frisians, who belong for the most part to the heavily-built blond race, are seldom musical. "Frisia non cantat." The Teutons regard the Italians, the Magyars, the Jews, and the Gypsies as musical, and rightly so. The South Germans are, in general, more musically inclined than are the North Germans, and I ascribe this to there being among the South Germans a lesser admixture of the heavily-built blond race, and a larger admixture of the Near Eastern race.

Of the "Dinaric race", Günther writes: "Their mind seems more parsimonious than that of the Nordics, they are more settled in their homes, and their mental outlook is narrower; but their wills are as vigorous, they are quite as straightforward, and they have as strong a sense for the heroic. Their essence consists in rude energy and uprightness". I do not think that this description applies to the Bosnians, the Slovenes, and the Albanians, who are usually regarded as comparatively pure-blooded representatives of the "Dinaric race", but it does apply to the heavily-built blonds. A "rough but cordial tone" is characteristic of the Atlantic nature.

The racial endowments of the Teutons are often compared with those of the *Jews*, but those who make such comparisons are apt to overlook that neither of these groups can be regarded as a unified race. The Jews, however, are certainly more unified racially than the non-Jewish population of central

Europe, and this especially applies to their mental characteristics. Even in South Germany, although Near Eastern characteristics are by no means rare among the Gentile population, a Jew can usually be recognised at once by his bodily appearance. In North Germany it is hardly possible to mistake a Jew. But the mental racial peculiarities of the Jews are more marked even than the bodily, so that we might speak of the Jews as a "mental race". The fundamentals of the Jewish mind are those already described as characteristic of the Near Eastern race (see above, pp. 643 et seq.), but even more strongly developed.

I fancy that I am able to recognise the literary work of a Jew (scientific writings included) by the way in which the thoughts are developed and by the method of expression. Jewish authors seem to have a preference for quoting one another rather than Gentiles, and this may be mainly attributed to their finding the thoughts of their co-nationals more congenial.

The Jewish peculiarities could only have been produced by selection in the course of thousands of years, in a civilisation in which social life was highly developed. The process must certainly have begun and been carried a long way in Asia Minor, the homeland of the earliest high civilisations of the ancient world. But, since the diaspora, it is obvious that the selective conditions of the last two thousand years have continued to work in the same direction. Excluded from the labours of primal production (agriculture), not only by their own inclinations, but also in great measure by external forces, they naturally turned to commerce and similar occupations. The result was that, in the main, only those Jews could found a family who had special aptitude for acting as intermediaries in dealing with the goods produced by others, and in stimulating and guiding others' wishes.

If the peculiarities of the Jews are less conspicuous in the bodily than in the mental domain, this may arise from the circumstance that Jews whose bodily aspect was markedly exotic were less successful than those whose bodily type resembled that of their hosts. The instinctive desire not to look singular would also operate by sexual selection, by a preferential choice of a partner in marriage who did not look too much unlike the hosts.[1] The adoption of Gentile names by many Jews is likewise a manifestation of the wish not to be recognisable as Jews. Insofar as the type has become less conspicuous thanks to the working of such a process of selection, we have to do with the mimicry which is familiarly observed wherever a living creature gains advantages in the struggle for existence by acquiring a resemblance to some other organism. Thus we find different species of animals which are very closely alike, and which may be akin, but which are sharply distinguished each from the other in matters of instinct. Among butterflies I know many such species. The great resemblance is here obviously due to the fact that the mimicry has been advantageous to one of these species in the struggle for existence.

We can understand, therefore, why it is that the Jews are characterised, not only by shrewdness and alertness, by diligence and perseverance, but also by an amazing capacity for putting themselves in others' places (empathy) and for inducing others to accept their guidance. Both by patience and by talent, therefore, they always incline to engage in occupations in which regard for the extant leanings of the public and the guidance of the public taste bring success. Consequently the professions they especially choose, and practise with advantage to themselves, are those of merchant, small trader, money-lender, journalist, author, publisher, politician, actor, musician, lawyer, and medical practitioner.

Both in Europe and in North America, the clothing trade is mainly in the hands of Jews. Now, this is a kind of business in which success especially depends upon a capacity for influencing human beings. Changes of fashion are prescribed by Jews for men and for women alike. The theatres are for the most part in the hands of Jews; in the United States exclusively so, according to Henry Ford.[2] The same is true of the movies. A large proportion of daily newspapers and other periodicals are issued by Jews, edited by Jews, and provided with articles by Jewish journalists. The legal profession would almost seem to have been created specially for Jews. In oratorical ability and the arts of persuasion, the Jew greatly excels the Gentile. His volubility is in marked contrast to the Teutonic taciturnity, just as the "Jewish haste" contrasts with the quietude and the slow mobility of the Teuton. The internal aloofness from men and things so characteristic of the Teuton is hardly known to the Jew, who feels himself thoroughly at home among his fellow human beings. The Jew is more often importunate than the Teuton, and is also more sensitive: but even when he has been mortified by a repulse, he will usually return to the charge; for by nature he is eminently sociable.

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[1] Cf. Luschan, Völker, Rassen, Sprachen, Weltverlag, Berlin, 1922, pp. 168–169.

[2] Der internationale Jude, eleventh edition, Leipzig, 1923.

Werner Sombart has given a brilliant description of the Jewish talent for economic life.[1] A very large proportion of the mobile capital in Europe and America is in the hands of Jews. In banking and on the stock exchange their influence is decisive. In manufacturing industry, Jews are prominent as business managers, but rarely as the founders and conductors of great constructive enterprises. Among the manual workers engaged in industry, there are very few Jews. Where they form a large proportion of the population, as they do in Poland, and in New York city, we find many of them engaged in the minor handicrafts, tailoring being one of their favourite occupations. They are strongly averse to strenuous physical toil. We hardly ever find them engaged in agriculture. This cannot mainly be because they have been kept away from agriculture, for in the United States it has been open to them to become farmers, but there, likewise, they have preferred occupation as middlemen of one sort or another. The Jews do not seek out countries or regions where there is free land, but regions where busy people congregate.

In the Zionist attempt to resettle Palestine with Jews, it has been interesting to find that few of the settlers are conspicuously Jewish in type;[2] manifestly they are recruited for the most part out of the non-Jewish racial elements which have been incorporated among the Ashkenazic or eastern European Jews. Taking them all in all, Jews constitute only 3·6 % of the agricultural population of Palestine, and this percentage is declining.[3] Owing to their deficient talent or inclination for the primary work of production it would seem that a State system consisting exclusively of Jews would be impossible.

[1] Die Juden und das Wirtschaftsleben, Leipzig, 1911.

[2] R. N. Salaman, writing in the collective work Eugenics in Race and Faith, William & Wilkins Company, Baltimore, 1923.

[3] J. Brandt, Die jüdische Kolonisation in Palästina, "Archiv fur innere Kolonisation" vol. XVIII, 1926.

Where the Jews form only a small minority, their economic position is as a rule an extremely good one. Their influence and their power will be found to be much greater than is proportional to their numbers. Salaman shows this as regards England, and Ford as regards the United States; for Germany, much information relating to the matter will be found in Fritsch's *Handbuch der Judenfrage.*[1]

If we could select the most talented hundredth of the Gentile population of Germany, we should, of course, find them to be abler mentally than the German Jews, who likewise comprise about one hundredth of the population of the country; and we may presume that several more hundredths selected in succession for conspicuous ability from among the remaining Gentile population would still be abler than the Jews. But when we compare the average German Jew with the average German Gentile we cannot doubt that the Jews greatly excel in intelligence and alertness. In the higher schools, where the pupils represent a selection for talent, the proportion of Jewish children is many times as large as the proportion of Jews in the general population. In the elementary schools, where no selection has yet taken place, the Jewish children on the average shape much better than the Gentiles. Furthermore, in the Jewish pupils of the Vienna Commercial Academy there can be shown to exist a "better intellectual endowment" than in the Gentile pupils, although the Gentiles have naturally been subjected to precisely the same kind of selection as their Jewish fellow students.[2]

In a comparison between Jewish and Gentile children, we must, however, never fail to allow for the fact that the Jews are precocious. Comparatively common among them are "infant prodigies" whose intellectual powers are greatly in advance of their years. According to Salaman, however, the brilliant achievements of youthful Jews depend rather upon precocity and quick-wittedness than upon creative talent.[3] For this reason they seldom fulfil the promise of their early years. Still, Jews form an immoderately large proportion of undergraduates at the universities. At the Prussian universities during the year 1911–1912, according to Fritsch, there were:

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[1] Twenty-ninth edition, Leipzig, 1923. Unfortunately this book is unduly polemical, and therefore too little objective. In a sense it may be said to be unduly "Jewish", and not "German" enough.

[2] O. Nemecek, Zur Psychologie christlicher und jüdischer Schüler, "Beiträge zur Kinderforschung", no. 128, Langensalza, 1916.

[3] Op. cit., p. 138.

	per 100 undergraduates	per 10,000 male inhabitants
Protestants	66·4	13
Catholics	27·5	5
Jews	5·6	67
Others	0·6	6

In the world of knowledge, the Jews are much more strongly represented than might be expected from their number. Their percentage among investigators and discoverers is, indeed, not so high as their percentage among teachers and men of learning. On the whole, therefore, it is indisputable that in the domain of mental life, too, they are more prominent as intermediaries and interpreters than in the primary work of production. On the other hand, it is absurd to deny that the Jewish race has produced persons of outstanding genius. Enough to mention the names of Spinoza and Einstein!

Spinoza was a true philosopher. Chamberlain has done him injustice. To Spinoza we can aptly apply the words used by Nietzsche in his delineation of the ideal man as conceived by Schopenhauer: Spinoza took upon himself the voluntary passion of sincerity. The Jews cast him out and cursed him; and even the Christians avoided intercourse with him, as if he had been the black sheep of the flock. Nonetheless, he remained true to his convictions, and maintained his spiritual independence. He made his own livelihood in the labour of primal production, namely as a lens-grinder. Since his family came from Spain, we may suppose him to have been rather of the Oriental than of the Near Eastern race. Einstein, likewise, would seem to be mainly of Oriental type.

The Jews have produced noted scientists in the domains of physics, mathematics, medicine, and psychology. In physics and mathematics, their successes have been rather in the abstract than in the concrete field; here their strength lies in their highly developed sense for numbers, and in their gift for formal logic. It is to the same heredity factors, doubtless, that the Jews owe their remarkable skill at chess. Most of the great masters of this game have been Jews.

Jews show comparatively little interest in concrete natural objects, their chief concern being with all the factors of mental activity. Most of those who have given special attention to the problem of the sexual life have been Jews. Freud, the founder of psychoanalysis, can be regarded as a typical Jewish thinker;[1] and so can Adler, the founder of individual psychology.

In the medical sciences, Jews are especially conspicuous in the fields of venereal disease, diseases of children, and diseases of the nervous system, whereas the Teutons display a contrasted aptitude for surgery. The field of social hygiene has been mainly worked by Jews. The fondness of the Jews for the art of healing dates from classical times. In part this devotion to medicine may doubtless arise from the circumstance that the Jew dreads pain, illness, and death, more than does the Teuton; but a powerful factor may also be that the success of the physician so largely depends upon his capacity for exerting a mental influence over his fellow human beings.

The physical sciences apart, the scientific fields preferred by the Jews are economics, philosophy (especially in its psychological aspects), the history of art, the history of literature, and Germanistics.

I have already alluded to the fact that a disproportionally large number of great musicians have been Jews. (See above, p. 646.) On the other hand, very few distinguished painters have been Jews, and scarcely any great sculptors or great architects. The visualising and technical ability of the Jews is comparatively small. The faculties of ear and tongue are in them more developed than the faculties of the eye. In the selection of the Jewish physique, too, a sense of form would seem to have played comparatively little part. Modern expressionism, to which the Jews have contributed much, is rather an art of mental expression than an art of configuration—a quasi-musical gift.[2] No doubt the Jews play a decisive part in the trade in objects of art, but they owe their position here to their instinct for what will please their fellow human beings and to their capacity for influencing the popular taste.

[1] Cf. A. Kutzinski, Sigmund Freud, ein jüdischer Forscher, "Der Jude", vol. VIII, 1924, no. 4.

[2] H. Berl, Die Juden in der bildenden Kunst der Gegenwart, "Der Jude", vol. VIII, 1924, nos. 5 and 6.

Their marked imaginative insight (empathy), their power of putting themselves in others' places, makes the Jews born actors. Famous Jewish actors have been, for instance, Kainz (born Kohn), Sarah Bernhardt, Moissi, Possart, Reinhardt (born Goldmann), Moscowitch. The Jewish aptitude for expression by words and gestures is also helpful to them as actors.

The Jewish talent for living among purely imaginary ideas as if they were concrete facts is advantageous, not only to the actor, but also to the barrister, the trader, and the demagogue. When a barrister can persuade himself that his client is in the right, when a trader can persuade himself that his wares are unrivalled for their excellence and their cheapness, when a party politician can feel positively convinced that the doctrines of his own party are the only reasonable and sound ones, these persons will in practice be far more convincing than if they retained a purely scientific appreciation of the concrete facts. A genuinely felt pathetic vein, the ability to pull out the emotional stop, are also extremely useful here. In revolutionary movements, hysterically predisposed Jews play a great part, being able to give themselves up unreservedly to utopian ideas, and therefore able with a sense of inward sincerity to make convincing promises to the masses.

To name two great men of an earlier generation, both Marx and Lassalle were Jews. In our own time, among prominent revolutionists, names that at once occur to our minds are those of Eisner, Rosa Luxemburg, Leviné, Toller, Landauer, Szamuely, and Trotsky. F. Kahn[1] extols these Jewish revolutionists as the saviours of mankind, discerns in them "the specifically Jewish variety of philosophy and of historical activity". Many anti-Semites believe that Jews are by their very nature inclined towards negation and destruction. I cannot agree with this view. The fact that so many of the leaders of revolutionary movements have been Jews is, rather, to be explained much more simply, as an outcome of the peculiarities above described, peculiarities which by no means tend towards destruction. Even when the Jew destroys, it is usually with the aim of rebuilding.

The strong family sense of the Jews is anything but destructive; and the same may be said of their strong sense of tribal interdependence, their readiness to render mutual aid, and their feeling for humanity at large. Next to the Teutonic, the Jewish spirit is the chief motive force of modern western civilisation.[1] The emancipation of the Jews has had an effect like that of one of the waves of Nordic blood upon the Indo-Germanic civilisations. Were it merely through the origination and diffusion of Christianity as one of the main roots of western civilisation, the Jewish spirit has been decisively effective in universal history.

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[1] Die Juden als Rasse und Kulturvolk, third edition, Welt-Verlag, Berlin, 1922.

The sobriety of the Jews, their power to resist the seductive charms of alcohol, might set an example to the Germans. Obviously their sobriety is based on their hereditary mental endowment, whereas the Teutons in this respect manifest a weakness of which a part cause, unfortunately, is beyond question their racial heritage. But that this trouble is not ineradicable, we may learn from the examples of Sweden and the United States of America.

Extremely interesting is the fondness of the Jews for Lamarckism, that is to say for the doctrine of the "inheritance of acquired characters". The advocates of Lamarckism are mainly, and the opponents of Lamarckism only to a very small extent, of Jewish descent. Hand in hand with this there usually goes a dislike of Darwinism, of the doctrine that the origin of species has been effected by the "cruel" process of natural selection during the struggle for existence, and not by way of the "peaceful" inheritance of acquired characters. During the last few years, there has been a change in the picture, inasmuch as Lamarckism has lost ground among the faithful, with the result that there are somewhat more Jews than there used to be among its opponents.[2] The inclination of the Jews towards Lamarckism is obviously an expression of the wish that there should be no unbridgeable racial distinctions. For instance, it is extremely characteristic that Kammerer, who was himself both a Jew and a Lamarckian, should write: "The denial of the racial importance of acquired characters favours race hatred." I am personally acquainted with Jews of high mental attainments who feel themselves to belong to the German people and to German civilisation, and to whom, therefore, it is the great tragedy of their lives that they should be looked upon as aliens. If acquired characters could be inherited, then, by living in a Teutonic environment and by adopting a Teutonic culture, the Jews could become transformed into genuine Teutons. This enables us to understand why the Lamarckian doctrine should make so strong an appeal to the Jews, whose fate it is to exist everywhere among the Gentiles as a sharply differentiated minority. But this, of course, can make no difference to the fact that the Lamarckian doctrine is an illusion.[1] In this connexion, Kahn writes aptly: "Jews do not transform themselves into Teutons by writing books about Goethe."[2]

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[1] This sentence in the first edition of the present work aroused the anger of anti-Semites among my readers, and gave rise to acrimonious criticism. I continue, however, to regard the statement as sound. It is a pity that so much enthusiasm and youthful energy should waste itself in the idle clamour of the anti-Semitic movement. If only this youthful energy could be canalised where it would be of real advantage to our race! On the other hand, I really must warn my Jewish fellow citizens that they ought not to get the wind up as soon as any one begins to speak about the Jewish race. This inevitably arouses the impression that they must have some reason for fighting shy of any exposition of racial questions. Yet a tranquil and objective discussion of the Jewish problem would best serve the true interests of both sides.

[2] The leading advocate of Lamarckism, Paul Kammerer, committed suicide in the year 1926, after G. K. Noble ("Nature", August 7, 1926,

Some writers, among whom may be mentioned the late Natorp, sometime professor of philosophy at Marburg, have declared that the peculiar characteristics of the Jews (insofar as their existence is not flatly denied) are an outcome of the oppression they endured during the ghetto epoch. The implication here is that the peculiarities of the Jewish mind are blameworthy, and the blame is assigned to the Gentiles among whom they have lived. Thereupon the inference is drawn that the Jews must not only be unreservedly accepted into the community life of the nation, but must be received

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p. 209) had proved that certain dark markings in the skin of the accoucheur toad, which Kammerer adduced as proof of the inheritance of acquired characters, were nothing of the kind, but had been brought about by the injection of Indian ink. Prior to this, Megušar, one of Kammerer's collaborators with whom he had fallen out, declared that he had never been able to accept any of Kammerer's alleged proofs of the inheritance of acquired characters, although he had been watching Kammerer's breeding experiments for ten years.

[1] The Jewish Lamarckian writer Iltis in his book on Mendel (Berlin, 1924) misrepresents me as having said that I had given the Lamarckian doctrine its quietus by simply pointing out that the majority of the Lamarckians are Jews. I need hardly say that I have never drawn any such inference. Lamarckism has received its quietus for very different reasons. I drew attention to the fact that most of the Lamarckians are Jews, not in order to give the mercy stroke to Lamarckism, but in order to illustrate the psychology of the Jews.

[2] F. Kahn, op. cit.

with exceptional tenderness and affection. Obviously this, likewise, is but Lamarckian illusion. The peculiarities of the Jews are not a direct product of their environment, but, as I have shown above, are the outcome of natural selection in the conditions under which the Jews have lived for thousands of years.

The disinclination of the Jews for war can also be explained as an outcome of selective processes. When the ancient Hebrew State was being politically subjugated by its more powerful neighbours, the most pugnacious among the Jewish families were wiped out, as happened, for instance, in the sanguinary struggles of the Maccabees against the Ptolemies. On the other hand, the more peaceable and obedient sections of the population remained alive to reproduce their kind. In the days of the Roman empire, when the Jews who stayed in the East had engaged in several revolutionary risings, a very large proportion of the Jews who yet remained in Palestine were slaughtered during the repression of these revolts by Titus, Trajan, and Hadrian. Before this, however, hundreds of thousands of Jews had been diffused throughout the Roman realm as traders, as the body slaves of well-to-do Romans, and as persons engaged in other occupations necessitating adroitness in social intercourse—and the Jewry of modern times has been mainly the outcome of this peculiar kind of selection. The savage persecutions to which the Jews were exposed during the Middle Ages and which have persisted even into our own day, have continued to work in the same direction as a selective force.

Although for the purposes of the present argument, the differences in hereditary endowment between the Jews and the Teutons have been brought into the limelight, the reader must on no account forget that in respect of many heredity factors Jews and Teutons are extremely alike, especially if we limit the application of the term "Teutonic" to persons of a predominantly Nordic race. Jews and Teutons are alike distinguished by great powers of understanding and by remarkable strength of will; Jews and Teutons resemble each other in having a large measure of self-confidence, an enterprising spirit, and a strong desire to get their own way—the difference being that the Teuton is inclined to seek his ends by force, the Jew rather by cunning. As far as business efficiency is concerned, the Jews are, on the whole, excelled by some of the Nordics, such as the Hanseats, the Scots, and the Yankees. The frequent outbreaks of enmity between Germans and Jews may be due, and probably are due,, not only to essential differences between the races, but quite as much to the similarity in their respective gifts, which lead to a strong competition like that which has again and again led the ruling groups of the Teutons into conflict one with another, each striving to establish power over the others. The Teutons of Nordic descent are likewise inclined to diffuse themselves as a ruling caste over foreign populations. They, too, prefer, whenever they can, to have the hard physical toil of life done for them by others—for that is a universally human characteristic, and differences in this respect are only differences of degree.

There is another thing in which the Teutons might take example from the Jews, namely, the latters' sense of mutual fellowship. The collaboration of the Jews goes so far that many anti-Semites actually believe (falsely, beyond a doubt) that there is a universal secret conspiracy of Jews. If Jews are more inclined to render mutual help than Gentiles are, this must be mainly dependent upon the peculiar position of the Jews.

Judophobia, usually but less aptly spoken of as "anti-Semitism", is, one may suppose, for the most part the outcome of a feeling of insubordination. "The Jews form an upper stratum, the Germans—the opposite", writes Fritsch. The spirit which animates many of the anti-Semites is the "envy felt by a non-possessing class". Superadded to this is a feeling that the Jews owe their advantageous economic and social position, not so much to their own useful and concrete activities, as to a special capacity for exploiting the achievements of others. At work, here, is the psychology of the Marxian "doctrine of surplus value". But it would be wrong to suppose that the Jews are merely parasitic. There can be no doubt that they contribute to the primal work of production by stimulating needs and by intermediating in the satisfaction of these. In this way they help economic life to thrive. Still, it is true that whereas the Teutons could get along fairly well without the Jews, the Jews could not get along without the Teutons. Moreover, it can hardly be agreeable to reflective and far-sighted Jews, that Jews should make the peculiar contribution they actually do make to the "spiritual life of the nation". Unfortunately by far the greater part of the literature relating to Jewish peculiarities is unmistakably tendentious. The Jewish question arouses such strong feeling, that those who write about it are wont either, like Chamberlain, Fritsch, and Ford, to take sides against the Jews, or like Zollschan, Hertz, and Kahn, to espouse the Jewish cause with passion. The result of such a bias is to impair an author's judgment, and to make him lack objectivity in the choice of his data. Even so, the writings in question can still be useful sources of information.

The frequency with which the members of various races are guilty *of criminal acts* throws some light on the mental differences between these races. In the United States, there is much more criminality among the Negroes than among the immigrants from Europe and their descendants. This is obviously due to the fact that the Negroes have less foresight, and that they have less power of resisting the impulses aroused by immediate sensuous impressions. No doubt the comparatively unfavourable economic position of the Negroes is a contributory cause, but this is in its turn referable to the peculiarities of the Negro racial endowment. It follows that the effects of racial endowment in producing minor and major offences are predominantly indirect rather than direct. In the United States, there is a much higher criminality rate among the immigrants from the southern and eastern parts of Europe than among those from the north-western half of Europe.[1] In the countries of Europe which are predominantly inhabited by Nordics, life and property are much more secure than in the southern countries of the European continent. The traveller in the lands bordering on the Mediterranean must be continually on his guard against being cheated or against having his possessions stolen; but in the northern parts of continental Europe, and also in Great Britain, he can with considerable confidence trust himself and his possessions to the honour of the population. It is difficult to see what there can be but difference in race to account for these differences in behaviour. Self-control, foresight, self-respect, and respect for the life and property of others, go a long way to prevent the Nordics from committing offences against the law.

An additional reason for the comparative infrequency of crime in Nordic countries would appear to be that the Nordic is disinclined to bother himself about trifles. Frauds on the large scale are by no means uncommon in Scandinavia and in England. In great enterprises, the Englishman is often rather unscrupulous, just as he is in politics. In the Americas, the security of life and property is much greater in the United States than it is in Central and South America. In politics, on the other hand, the United States citizen is apt to be ruthless in pursuit of his interests.

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[1] A. Ploetz, Sozialanthropologie, a contribution to the collective work Anthropologie, Teubner, Leipzig, 1923.

Lombroso[1] was one of the first to draw attention to the fact that in Europe murder and manslaughter were least common among the Teutonic peoples, and most common among the Latin peoples; and that in Italy these crimes are commoner in the southern part of the peninsula and upon the islands than in northern Italy.

It would seem, then, that the Mediterranean race is comparatively prone to commit these offences. In Sardinia, murder and manslaughter are fourteen times as common as they are in Lombardy.[2]

In Germany, the frequency of crime in the north and western parts of the country is much less than in the eastern and southern parts. During the years 1882 to 1891, per 100,000 persons of a legally punishable age, crimes and misdemeanours were committed to the following extent in different parts of the country:

Hanover, Oldenburg	711
Hesse-Nassau, Grand-Duchy of Hesse	729
Rhenish Prussia	746
Wurtemberg and Baden	811
Pomerania, Schleswig-Holstein, Mecklenburg	822
Silesia (without Upper Silesia)	1060
Bavaria (without the Palatinate)	1170
East Prussia and West Prussia	1570
Posen	1612
Upper Silesia	1711

The southern areas occupied a middle position, and throughout the German realm as a whole there were rather more than 1000 offences per 100,000 inhabitants.

It would seem, then, that the heavily-built blond race is just as little inclined to crime as is the slender blond race. Whereas speaking generally the Catholics in Germany have a 20 % higher criminality rate than the Protestants, Münster, though a Catholic region, is (as Pollitz has pointed out)[3] distinguished for the rareness of crime. Obviously, then, these differences in the frequency of crime must depend mainly upon racial differences, whereas religion, as such, seems to have very little to do with the matter. Unfortunately, in German criminal statistics since the year 1901, no mention has been made of the offender's religion, so that only the earlier figures are of any use to us here. In the decade 1892–1901, in the German Empire, the average number of offenders per 100,000 persons of a punishable age was 1207. When offenders are classified by religion, we get the following results:

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[1] L'uomo delinquente, fifth edition, Turin, 1897.

[2] Cf. G. Aschaffenburg, Das Verbrechen und seine Bekämpfung, third edition, Heidelberg, 1923.

[3] Cf. G. Aschaffenburg, op. cit.

	Catholics	Protestants	Jews
offences in general	1361	1112	1030
minor assaults	67·0	53·7	44·0
grievous bodily harm	314·1	185·5	75·3
fraud	68·2	57·5	112·8

The greater frequency of assaults among Christians than among Jews is, presumably, due not to religion but to race; and the difference in this respect between Catholics and Protestants must likewise be mainly due to the fact that German Catholics and German Protestants differ in respect of race.

In the matter of theft, too, the Jews occupy a better position than the Christians (in the case of minor theft, 80 as against 231, and in the case of major theft, 11 as against 33); but an unfavourable position in the matter of the use of insulting language (200 as against 143), fraud (113 as against 61), and forgery (25 as against 13). Of course differences in social position and above all in occupation play a great part in accounting for these differences. On the other hand, racial distinctions are partly responsible for differences in social position and in occupation. G. Aschaffenburg[1] also believes that the racial endowment of the Jews finds expression in the nature of the offences they commit. The fact that they are comparatively little inclined to deeds of violence is in large measure to be referred to their moderation in the use of alcoholic liquors, but this in turn depends upon their mental peculiarities. If we are to reach an objective judgment concerning the criminality of Jews, we must compare Jews, not with the non-Jewish population in general, but with those sections of the non-Jewish population which occupy a similar social position to the Jews. My own impression is that when this is done it will be found that deeds of violence are no more frequent in the non-Jewish than in the Jewish population, but that fraud and the use of insulting language really are commoner among Jews.

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[1] Op. cit.

It is said that Jews are especially responsible for the circulation of obscene books and pictures, and for carrying on the White Slave trade. Most of the White Slave traders are said to be Ashkenazic Jews.[1]

In any case, there are close relationships between race and crime. I am inclined to think that there is even a certain amount of truth in the doctrine formulated a good many years ago by Lombroso, that the "born criminal" belongs to a special and primitive racial stock. Criminals very often exhibit characters which remind us of Neandertal man or of other primitive races, having prominent and massive jaws, receding foreheads, etc. When a race is for the most part exterminated, or driven out, by another, there is nevertheless almost invariably some mingling of the bloods, so that elements of the hereditary equipment of the suppressed race continue to exist among the conquerors. It is, therefore, quite possible that vestiges of early diluvial man remain scattered here and there in the European population of to-day, and that the carriers of this primitive hereditary equipment are peculiarly apt to come into conflict with the demands of modern civilised life. We must also remember that no clear biological distinction can be drawn between the heredity factors of race, on the one hand, and other heredity factors including the heredity factors of disease, on the other. (See above, p. 623.)

In the subsection upon the hereditary insanities and psychopathies, an account was given of the relationships between varieties of mental disorder and varieties of bodily form (see above, pp. 461 et seq.); and in the section on the inheritance of intellectual gifts, an account was given of the relationships between talent, temperament, and character, on the one hand, and bodily characteristics, on the other. It remains, then, to consider what is *the nature of the relationships between mental peculiarities and bodily racial characteristics.* There can be no doubt that such relationships exist. The constitutional types distinguished by Kretschmer and others, form elements of the racial types. Thus the slender (leptosome) habit of body is characteristic of the Nordic race, and it is an undeniable fact that in the mental sphere the Nordic race is of "schizothyme" temperament, in Kretschmer's sense of the term. The stocky (pyknic) habit of body is typical of the Mongoloid race and also of the Near Eastern race; and here, likewise, the mental disposition harmonises with the bodily one inasmuch as the "cyclothymic" traits which Kretschmer has described as prevailing among pyknics are also conspicuous in Mongoloids and the Near Easterns. The athletic habit of body is characteristic of the heavily-built blond (Atlantic) race;[1] and here also we find that Kretschmer's observations concerning the schizothyme mental equipment of the athletic type is in conformity with the general mentality of the heavily-built blond race. The Mediterranean race, whose members are small in build and of a restless disposition, is distinguished from the Atlantic race, whose members are tall and of a thoughtful disposition, much as a small and restless terrier is distinguished from a great and reposeful St. Bernard. For our present purposes, we have no concern with the constitutional types classed by Kretschmer as of the dysplastic type, for these are the outcome of morbific heredity factors, and have no bearing upon the normal differences between the great races of man.

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[1] Cf. E. Wulffen, Der Sexualverbrecher, ninth edition, Langenscheidt, Berlin, 1922.

It has long been noted that hunting and sea-faring stocks are generally slender and long-headed, whereas settled peoples who make their living as agriculturists and handicraftsmen are generally stocky and broad-headed. There must obviously be correlations, on the one hand, of slenderness of build and narrow-headedness with a roaming disposition and, on the other, of a stocky build and broad-headedness with plumpness and a disposition to settle down.[1] The slender types are found to predominate, first of all, in populations which have been mainly recruited by immigration (as in the United States) and, secondly, in the upper social strata, which represent a selection of comparatively mobile racial elements. The stocky type, on the other hand, predominates in what are called backward areas (mountain regions, etc.), and also among the lower classes of society. Now that attention has been drawn to these facts, the slender type has become fashionable, and a great many young ladies endowed with a pyknic constitution have had to live on spare diet. Of course mental alertness is not directly dependent upon the bodily constitution and the shape of the head; for we must suppose the association between the two to be largely determined by hormonal influences, which, in turn, depend upon hereditary predisposition (see above, pp. 463 and 488). L. Stern-Piper [2] has, indeed, accurately recognised the connexion between race and bodily constitution, but has explained that the connexion exists—that a correlation between habit of body and certain hereditary mental trends exists—because every race has its own mental peculiarities. We have to remember, however, that the type of a great race is not transmissible by inheritance as a unit. What is hereditary is each individual racial heredity factor taken by itself. If, nevertheless, in mixed populations likewise, we find relations between the bodily type and the mental, there must be certain genes upon which distinctive bodily and mental peculiarities simultaneously depend. We are, therefore, entitled to say that, inasmuch as to the type of a race there belongs a particular bodily constitution, which is associated with a particular mentality, the great races must have the mental traits appropriate to their bodily constitution. Stern-Piper has inverted this relationship. All the same, he has got much nearer to the truth than have his critics. K. O. Henckel[3] has found that within a population the leptosome bodily constitution is not correlated with fairness of tint nor the pyknic bodily constitution with darkness of tint; and therefore he and also F. von Rohden[1] infer that "the constitutional habit of body is independent of the racial habit". Thus these authors set out from the erroneous assumption that the various heredity factors of a race must be correlated in a mixed population also. Pearson, however, showed a good many years ago that the tint of eyes and hair in the English population was nowise correlated to stature or to the shape of the head; and recent genetic research does not lead us to expect that we shall as a rule find a correlation between the various heredity factors of a race. The erroneous reasoning of Henckel and Rohden would, if carried to its logical conclusion, lead us to infer that every hereditary character must be independent of race. Yet it is obvious that every race has a typical bodily form. We must, therefore, reject as untenable Henckel's statement: "The same constitutional forms, the leptosome, the muscular, and the pyknic bodily types, are found in all the European races." They are only found *interspersed* in all the European races. If any one has in other respects all the characters of the Nordic race, but has a pyknic bodily constitution, he cannot be regarded as a pure-blooded representative of the Nordic race. Besides, it must not be supposed that the various bodily types are equably distributed among the various racial mixtures. For instance, the leptosome type is far more widely diffused in Scandinavia and in England than in central Europe or in Russia. At my suggestion, A. Verschuer[2] restudied as to origins the data he obtained by measuring Tübingen students. It then became apparent that among the students from southern Germany only 24 %, whereas among those from central Germany and northern Germany about 43 %, were of leptosome type. Now, the heavily-built blond race, which forms the foundation of the population of North Germany, is not of a leptosome habit of body; but still, the slender blond (Nordic) race is much more strongly represented in North Germany than in South Germany, and in conformity with this the leptosome habit of body is much commoner in North Germany. Noteworthy, too, is the fact that Verschuer was able to show that among the Tübingen students, "the leptosome type was much the commonest", although this is by no means the case in the general population of Wurtemberg. The distribution found by Verschuer is obviously the expression of a social selection, whereby the leptosome type is accumulated in the upper classes.

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[1] F. Paudler, Die hellfarbigen Rassen, Heidelberg, 1924, p. 167.

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[1] The reader may be referred to D. R. Stockard, Human Types and Growth Reactions, "American Journal of Anatomy", vol. XXXI, 1923, no. 3; R. B. Bean, The Two European Types, "American Journal of Anatomy", vol. XXXI, 1923, no. 4.

[2] Kretschmers psycho-physische Typen und die Rassenformen in Deutschland, "Archiv für Psychiatrie", vol. LXVII, 1923, no. 5.

[3] Ueber Konstitution und Rasse, etc., "Zeitschrift für Konstitutionslehre", vol. XII, 1926, no. 2.

If the normal schizothyme equipment is more widely diffused in the northern parts of Europe, this does not mean that the morbid predisposition to schizophrenia is necessarily more prevalent in these regions. The diffusion of morbific heredity factors depends mainly upon processes of selection, the existence of such factors being restricted by the degree to which they tend to impair the chances of survival. Henckel insists that schizophrenia is no commoner in Sweden than in other countries. But all that his figures denote is that in Sweden manic-melancholic disturbances are comparatively rare. In his Swedish material there were 49 manic-melancholics to 350 schizophrenes, that is to say a proportion of 1 : 7; but in his Upper Bavarian material there were 73 to 100, that is to say a proportion of 1 : 1·4. In Kretschmer's Wurtemberg material, the ratio was 85 : 175 = 1 : 2·2. In Russia, where these two groups of mental disorder are separately tabulated, Rohden found in the year 1922 a ratio of 12 % : 28 % = 1 : 2·4. It seems to me that these figures indicate that in central and eastern Europe, where pyknic types are much commoner than they are in Sweden, manic-melancholic disorders are comparatively frequent. If, nevertheless, schizophrene disorders are no rarer there than in Sweden, this may be determined, apart from other selective conditions, by the races being more mixed. Henckel, indeed, believes that this possibility can be excluded, inasmuch as in his schizophrenes he could not detect any disturbance of the correlation between the tint of the hair and the tint of the eyes. However, this conclusion likewise is based upon his erroneous initial assumption that the heredity factors which are correlated in a primary race must be correlated in the same way in a mixed population. In actual fact, the correlation between the tint of the hair and the tint of the eyes depends rather upon this, that particular heredity factors simultaneously determine the pigmentation of the hair and the pigmentation of the eyes. We have not as yet found any evidence to exclude the possibility that a mingling of races may lead to the production of types that are also dysplastic and disharmonious in respect of their bodily characteristics.

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[1] Ueber Beziehungen zwischen Konstitution und Rasse, "Zeitschrift für die gesamte Neurologie und Psychiatrie", vol. XCVIII, 1925, nos. 1 and 2.

[2] Zur Frage Körperbau und Rasse, "Zeitschrift für Konstitutionslehre", vol. XI, 1925, no. 6.

From the circumstance that racial factors do not maintain their correlation in a mixed population, it follows that we cannot, in such a mixed population as our own, simply infer an individual's bodily characters from his mental racial endowment. It is, for instance, possible that a man with light-coloured eyes and fair hair may have a mental constitution which properly belongs to a dark race. With somewhat more probability, on the other hand, than from such bodily racial characters, we can infer from a man's origin what his mental racial endowments will be. Among the peasants of Lower

Saxony, small, short-headed, dark-haired persons are quite exceptional, but, all the same, it is far more probable that a Lower Saxon will have a Nordic mental constitution than, for example, that a tall, blond, long-headed Italian will be of this mental type.[1] Since most persons, however, do not constitute exceptions of the above-mentioned kind, but are endowed with the average bodily and mental qualities of the groups to which they belong, we can often with considerable accuracy infer the mental constitution from the bodily type; and in practical life we are all in the habit of doing this, more or less.

The idea that the various heredity factors of a race must necessarily be correlated, presumably derives from the period when people still believed that the type of a race is transmitted by heredity as a unit; or when they believed, as Weismann imagined, that every heredity unit contained the factors for the entire organism. Although we know now that this is not the case, it does not follow that the racial endowments have no bearing upon an individual's mental peculiarities. What is essential, however, is that we should accustom ourselves to the idea that persons outwardly resembling one another in type may have different mental equipments, and that persons differing from one another outwardly in type may have like mental endowments. Furthermore it does not follow that two persons will be alike in respect of racial type because both of them derive their hereditary equipment in equal proportions from particular races. What matters is, which particular factors they have respectively acquired from the different races. In like manner two different populations which represent racial admixtures in identical proportions are not necessarily themselves racially identical. Owing to the selective effects of environment, in one of these two populations some of the heredity factors of the primary races, and in the other population other heredity factors of these races, may have been preserved.

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[1] The foregoing sentence was worded exactly as above in earlier editions. Nevertheless, K. F. Wolff (in his Rassenlehre, Leipzig, 1927) misrepresented me as saying that I had "ascribed all possible evil qualities to the short-headed dark Germans". W. Pfuhl, likewise (in his Die Beziehungen zwischen Rassen- und Konstitutionsforschung, "Zeitschrift für Konstitutionslehre", vol. IX, 1923, no. 2), has misunderstood me when he writes: "Lenz proposes to distinguish two different races simply by the characters fair and dark." What I wanted to do, on the contrary, was to insist upon the independence of pigmentation from the other racial peculiarities. To a very large extent, Pfuhl's views march with mine. Like him, I have drawn attention to the importance of the internal secretions in these matters.

We must clearly recognise that in a mixed population it is impossible to decide with certainty to which race an individual mainly belongs. Presumably all human beings have in common the greater part of their hereditary equipment; and it may well be that racial differences are determined by no more than a very small proportion of the heredity factors—in a word, that the greater part of our hereditary equipment has nothing to do with racial distinctions. From this it follows that there can be no possibility of ascertaining the share of different races in an individual's hereditary equipment. Still less is it possible to decide to which race a person belongs from the study of a few of his external characters, inasmuch as these do not warrant trustworthy conclusions concerning the rest of his hereditary equipment. Still, inferences having a fair measure of probability can be drawn. The problem is on the same footing as that which concerns the signs of talent (see above, pp. 600–1). The indications of talent and of character may be either indirect or direct. The indirect indications may be due to the fact that in a particular environment certain mental qualities and certain bodily qualities may both be favoured by selection, although there is no direct connexion between the bodily qualities and the mental. In this way, however, there will arise a measure of correlation between mental and bodily characteristics, just as such a correlation arises between two or more bodily characteristics. When we are considering large countries or entire continents, we can, with considerable probability, infer that the presence of certain bodily characters will entail the presence of certain other bodily characters. When we are studying Europe as a whole, and still more plainly when we are studying the continents of Europe and Asia taken together as a whole, we find, for instance, a marked correlation between the tint of the eyes and the shape of the nose, although in small areas we cannot detect any such correlation. Dealing with the human race as a whole, we can with a high degree of probability infer that an individual with frizzy hair will have a dark skin; and, in like manner, we can with considerable probability infer that certain bodily characters will be accompanied by particular mental traits.

But certain bodily racial characters appear also to warrant direct conclusions regarding mental endowment. Thus the size of the forehead and the size of the head are to some extent racial characters, and we have shown above that mental gifts are associated with these bodily characters. Similar considerations would seem to apply to slenderness or stockiness of build, to the width of the face, etc. Presumably most of the heredity factors which determine an individual's mental equipment manifest themselves likewise by the presence of certain bodily characters which may be trifling and inconspicuous, and perhaps have no bearing upon selection. In that case they would owe their existence exclusively to "conselection", a process to which W. Scheidt ascribed great importance in the origination of racial characters.[1] I am inclined to restrict the notion of conselection somewhat more narrowly than does Scheidt, applying it only to those instances in which the selected and the conselected character are dependent upon the same gene. I must also remind my readers that racial differences mainly depend upon differences in the internal secretions.[2] Inasmuch as the effects of the internal secretions are of great importance in determining the outward bodily configuration, and inasmuch as their effects are also closely connected with the mental life, it should be possible on this ground to trace connexions between mental aptitudes and bodily racial characters, and to do this scientifically, whereas hitherto we have for the most part drawn such inferences instinctively and as the outcome of unconscious experience.

Often enough it is said that mental racial differences can be of no great importance because the mental differences within particular "groups" are more extensive than the mental differences between the different races of man. But here we have, first of all, to consider the signification of the term "group", which is often used in an extremely vague way. Apart from this, the statement is unsound, for the differences within the various linguistic groups (of which we think first in this connexion) themselves depend to a great extent upon racial differences. On the other hand, the "extent of variation" of the races is not a tenable standard of comparison—a fact that is to-day generally admitted in other departments of biology. For instance, that idiots are occasionally met with in all races is not a serious argument against the importance of racial distinctions.

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[1] Allgemeine Rassenkunde, Lehmann, Munich, 1925.

[2] J. Paulsen, Wesen und Entstehung der Rassenmerkmale, "Archiv für Anthropologie", vol. XVIII, 1921; A. Keith, The Evolution of Human Races in the Light of the Hormone Theory, "Bulletin of the Johns Hopkins Hospital", vol. XXXIII, 1922.

Another objection often voiced is that it is impossible to say with certainty to what race an individual belongs, so that all inferences concerning race must be uncertain. There is a fair amount of justification for such an objection as regards some of the data of ethnology. When, for instance, the heads of well-known persons are sketched or photographed, and are then produced as illustrations with the legend "Nordic" or "Ostisch", most readers will pigeon-hole them at once, with appropriate conclusions regarding the mental characteristics of the persons concerned. Yet this classification has often been effected in accordance with a very small number of characters, which may not warrant any inferences concerning the greater part of the hereditary equipment. Nevertheless the fact that such a procedure is unsound does not justify the conclusion that we are unable to form clear ideas concerning racial mental differences in general. Certainly the objection has no bearing upon the method followed in this book, in which we have deduced racial mental differences, not from individual types, but from the distinctions between extensive populations.

There is absolutely no justification for the view that anthropology must deal only with bodily characteristics. Of course mental qualities cannot be measured with a pair of compasses, but that is no reason for rejecting as unscientific all study of the mental differences of race. One who does this must, to be logical, also exclude all psychology and all psychiatry from the domain of science. Just as we can establish definite types of mental disorder, so can we establish racial mental types, and the latter distinction is certainly no less important than the former. Some of the scientists of the Lamarckian persuasion (see above, pp. 674–5), such as Franz Boas,[1] Luschan,[2] and others deny, not only the possibility of ascertaining racial mental differences, but even the very existence of such differences. All the mental differences between the various "groups of man" are to be regarded as the outcome of exercise, education, and other external influences; and the only reason why the "groups" of extant primitives have not developed a higher civilisation is that they have been living in unfavourable environments, and have not had the requisite "time" to become civilised. Such writers twist their arguments in order to show that all who maintain the existence of mental racial differences are prejudiced, whereas those who deny the existence of such differences are unprejudiced. Of course we who are not Lamarckians believe that the ascertaining of mental racial differences is a matter of practical importance, but it does not follow that everything which has practical importance is tendentious. Unquestionably tendentious, on the other hand, is the diffusion of ungrounded opinions in order to serve particular interests—and this applies to the denial of mental racial differences, a denial which runs counter to all practical experience and all reasonable consideration. No one can get out of his own skin or rid himself of his own mind, and minds are racially differentiated quite as much as are the tints of the skin.

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[1] Kultur und Rasse, Leipzig, 1914. (This is a German elaboration of the author's The Mind of Primitive Man, New York, 1911.)

[2] Völker, Rassen, Sprachen, Berlin, 1922.

Especially misleading is it, in this connexion, to sunder science into two sharply distinguished departments of the "mental and moral sciences" on the one hand, and the "natural sciences" on the other. Since Paul of Tarsus contrasted the children of Abraham in the "flesh" to the children of that patriarch in the "promise" [spirit], attempts have again and again been made, and especially in the nineteenth century, to restrict the natural sciences (biology included) to the physical domain. This has led to the erroneous but current view that only the body reproduces itself by means of the biological hereditary equipment, whereas the mind is supposed to reproduce itself exclusively by tradition. Such an identification of tradition with the mental hereditary equipment is untenable, as is the whole of the alleged contrast between "spirit" and "nature". The mind, too, has its laws, has a "nature" in Kant's sense of the term, and all natural science is an achievement of the mind. It is, therefore, utterly wrong-headed to contrapose "nature" and "spirit"; the decisive contrast of notions is, rather, hereditary equipment on the one hand, and environment on the other.

Connected with the problem of the mental peculiarities of the races of man, is the problem of the *effects of racial mingling* upon the mental endowments. Very general is the view that when races are mixed the mental constitution of the lower race invariably predominates, so that half-breeds are always inferior to either of the parental stocks. Thus we are told concerning the mixed populations of the coastal regions of Africa, Central America, and South America, that they are morally at a lower level than the pure-blooded Negroes and pure-blooded Indians from whose mingling they have sprung, although in intelligence they may be equal to either parental race or even superior to these.

B. Agassiz laid great stress upon the harmfulness of a mingling of races: "Let any one who doubts the evil of this mixture of races, and is inclined from mistaken philanthropy to break down all barriers between them, come to Brazil. He cannot deny the deterioration consequent upon the amalgamation of races, more widespread here than in any country of the world, and which is rapidly effacing the best qualities of the white man, the Negro, and the Indian, leaving a mongrel, nondescript type, deficient in physical and mental energy."[1]

Presumably the inferiority of these half-breeds is mainly the outcome of selection. The whites who enter into unions with native women represent an unfavourable selection as regards character; and, in like manner, the native women who enter into such unions with the whites must in many cases be of an unstable disposition. There can be no reason to wonder, then, that most of the half-breeds resulting from such unions should be of inferior type mentally and morally.

The half-caste or Eurasian offspring of unions between Europeans and Mongols in the Far East are described as being light-minded and happy-go-lucky. Here, once more, it is chiefly the light-minded individuals of both the racial groups who enter into such unions and procreate half-breeds. F. L. Hoffmann[2] reports that in his experience the individuals who in the southern States of the American Union enter into mixed marriages or illegal relationships between white and coloured, are far below the average of the two races. In the slavery days, on the other hand, this would seem to have been different, for then the prettiest among the slave girls were usually those who bore children to their masters.[3]

If the selection has been a favourable one, the half-breeds show no inferiority when compared with the coloured parental race. This applies, for instance, to the half-breeds of

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[1] Quoted by F. J. Holmes, The Trend of the Race, New York, 1921, pp. 248–249.

[2] Race Traits and Tendencies of the American Negro, New York, 1896; Negro-White Intermixture and Intermarriage, in Eugenics in Race and State, Baltimore, 1923.

[3] Holmes, op. cit., p. 263.

Rehoboth described by E. Fischer.[1] These were the offspring of Dutch colonists and Hottentot women who became the legal wives of the white settlers. Fischer found that these half-breeds were superior to pure-blooded Hottentots both in intelligence and in character, but that they were considerably inferior to the Boers in cultural endowments.

In any case there is no ground for the assertion that great cultural achievements are only possible to pure races, and still less ground for the contention that the purity of a race is more important than the nature of its racial endowment. The Nordic stocks which migrated into ancient Hindustan, Greece, and Italy, enjoyed their most splended flowering at a time when they had already incorporated manifold racial elements from the indigenous population. As in the bodily field, so likewise in the mental, there seems to occur a "luxurisation of hybrids". Many of the men who are universally regarded as the greatest in history (for instance, Socrates, Michelangelo, Luther, Goethe, and Beethoven) were obviously of mixed race. Speaking generally, it is exceptional to find that distinguished men exhibit a pure racial type.

F. Hertz is right when he says that "Most men of genius exhibit a mixed type";[2] but he draws an uncritical and prejudiced conclusion when he goes on to say that this fact is a striking refutation of the "illusion that racial minglings are harmful"—for genius is a rare phenomenon, and it might very well be true that in the generality of cases the effects of racial mingling would be unfavourable.

In my opinion there can be no doubt that the mingling of two races widely distinct each from the other may lead to the production of types which are disharmonious in respect alike of body and of mind. The particular heredity factors of every race have been mutually interadapted by natural selection through countless generations, and this harmony can be disturbed by mingling.

J. A. Mjöen[3] has reported some cases in which half-breeds of

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[1] Die Rehobother Bastards und das Bastardierungsproblem beim Menschen, Fischer, Jena, 1923.

[2] Rasse und Kultur, third edition, Leipzig, 1925.

[3] Harmonic and Disharmonic Race Crossings, in Eugenics in Race and State, Baltimore, 1923.

Norwegian and Lapp parentage were remarkable for immoral behaviour, whereas the parental families on both sides were well-conducted persons. Such isolated instances are of little value as proof, but we also lack evidence to show that Mjöen's view is unsound. H. Lundborg[1] likewise believes the crossing of races widely distinct each from the other to be deleterious, and so does O. Schlaginhaufen.[2] W. H. Cox[3] and others have advocated the view that a psychopathic constitution and even insanity may be the outcome of racial crossing; this seems possible rather than probable, and obviously it cannot be supposed that psychopathies originate in such a manner. Apart from this, the spread of morbific hereditary factors which have originated as mutations amounts to a disturbance of the racial purity.

The crossing of Teutons and Jews is likely, as a rule, to have an unfavourable effect, for it will impair the peculiar excellences of both types. Still, quite a number of celebrated men have been the offspring of unions between Gentiles and Jews,[4] as, for instance, the French philosopher Montaigne, the English astronomer Herschel, the Russian chemist Mendelejeff, the Russian biologist Metchnikoff, etc.—which, however, does not prove that these men of genius owed their gifts to the crossing of the races. It always remains possible that one or other of the parents might have had offspring no less remarkable in unions with persons of their own race. A very favourable mingling of blood would seem to be that between the slender blond (Nordic) race and the heavily-built blond (Atlantic) race. The Teutons, who have been the chief creators and are the chief sustainers of western civilisation, are the offspring of such a crossing. "The enormous expansion and great success of the Teutonic peoples bear unquestionable witness to a racial energy which is excelled nowhere in the world." Thus writes F. Hertz,[5] who is in general disposed to make light of racial differences, so his words here are all the more convincing.

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[1] Rassen- und Gesellschaftsprobleme in genetischer und medizinischer Betrachtung, "Hereditas", vol. I, 1920.

[2] Rassenmischung und Konstitution, "Natur und Mensch", 1921.

[3] Degeneratio, "Psychiatr. en Neurol. Bladen", 1907, no. 1 (a Dutch periodical).

[4] M. Fishberg, Intermarriage between Jews and Christians, in Eugenics in Race and State, Baltimore, 1923.

[5] Op. cit., p. 335.

People are inclined, following Chamberlain, to speak of Goethe as a salient example of a Teutonic or Nordic thinker, but I cannot agree with this view. Certainly Goethe had various Nordic traits, among the most conspicuous of which was his power of visual intuition; but many of his qualities were not Nordic. Consider, for instance, his dislike for hard truths and for clashes; his lack of understanding for tragical heroism; his amazing imaginative insight; his cosmopolitan sentiment; his fondness for making an imposing appearance; his ardent erotic fantasy, which is psychologically akin to the author of the Song of Solomon and to the mentality of the Near Eastern poets he so greatly prized—none of these are typically Nordic qualities. In more than one respect, Goethe seems blood-brother to Rousseau. His *Werther* is in Rousseau's vein, and yet the figure of Werther is not lifted bodily from Rousseau, but manifestly incorporates Goethe's personal experience as do many of his other characters. It seems to me most probable that Goethe's biological heritage was Near Eastern as well as Nordic. His bodily type and that of the members of his maternal kinship tend to confirm this hypothesis. R. Sommer considers that Goethe was "of mixed Teutonic and Latin type"; and he draws attention to the fact that in the Wetterau, to which the family of Goethe's mother belonged, there is "a marked mingling of Teutonic and Latin elements", and that "half-Latin or wholly Latin faces are often to be seen in the villages". What Sommer speaks of as a "Latin element" must rather, racially considered (and it is only with race that we have to do here), be not so much Mediterranean as Near Eastern. The Romans of the imperial age, from whom this admixture of "Latin" blood in the Wetterau derived, were in great part of Near Eastern descent; and furthermore the legions were accompanied wherever they went by Jewish and Syrian traders. The surname of Goethe's grandmother, Lindheimer, might lead us to suspect a comparatively recent and direct Jewish descent, but there is no evidence of this.[1]

The fact that Goethe must have been partly of non-Nordic origin becomes especially plain when we compare him with imaginative writers of an exclusively Nordic type of mind, such as Shakespeare, Schiller, Hebbel, Gobineau, Nietzsche, and Strindberg. These make no attempt to blink the terrible futility of existence; nay, rather, they seek to disclose it, finding therein a source of tragic elevation. Goethe, on the other hand, does not like the all-too-human. In his Faust he wanted to describe the fundamental motives of the human heart, and yet the eternal masculine is lacking there, despite all the talk about "action". Nietzsche, who as is well known was throughout his life a great admirer of Goethe, says of the generic man depicted by Goethe (whom Nietzsche compares and to some extent contrasts with the generic man of Rousseau and of Schopenhauer respectively): "He detests force, violence, sudden movement—which amounts to saying that he detests action." Napoleon's impression of Goethe was: "Voilà un homme qui a eu de grands chagrins!" (there is a man who has suffered much; ecce homo). Thus, what Napoleon saw above all in Goethe were the signs of suffering.

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[1] Dr. Walter Rauschenberger, Director of the Senckenberg library, has in the main accepted my views concerning Goethe, and has added several supplementary outlooks of his own. See his article Goethes Charakter und Abstammung, "Die Sonne", 1926, no. 9.

This heritage did nothing to diminish the greatness of Goethe's imaginative writing. On the contrary, we may say that if Goethe had had more sense for the eternal masculine, he would probably not have been so perfect a master of the eternal human. Presumably, therefore, the admixture of non-Nordic elements in his temperament was of immense significance to his work. He was greatest of all as a lyric poet, the only other German writer comparable to him in this respect being Heine. What I have wanted to illustrate by my account of Goethe's case is the fact that purity of race is not an indispensable prerequisite of mental superiority, and that, rather, some kinds of mental greatness are furthered by racial crossing.

Even a certain amount of discord among the various racial heredity factors is not necessarily unfavourable to the powers of cultural creation. Indeed, it has already been explained that positively disharmonious natures are sometimes peculiarly well fitted for great cultural achievements. Gobineau, to whom the apostles of the doctrine of pure race frequently appeal, himself declared that "the world of the arts and of the highest literature was an outcome of crossings of blood"; and he added, "it would be wrong to maintain that all minglings of races are necessarily evil and harmful". On the other hand it is true that he could not recognise any other race worthy to mingle with the Nordic, and it seemed to him to be the doom awaiting the highest race and the loftiest civilisation that the sustainers of that civilisation, being comparatively few in number, would gradually have their nobler blood diluted and debased by the floods of the ignoble.

The opinion often voiced by the disciples of Gobineau and Chamberlain that classical civilisation collapsed because of the mingling of the races cannot be maintained in any such narrow form. The decisive factor in the collapse of the classical world was, not a mingling of blood, but the extermination of the creative racial elements. This matter will be discussed more fully in the sequel to the present volume.

The reason why the peculiar cultural endowments of certain races are reported with so much emphasis by some observers and so passionately denied to exist by others is that people believe the value of a race to be dependent upon its cultural gifts. For instance, A. Grotjahn[1] writes: "The only objective criterion of the value of a race is contained in its cultural achievements, as recorded in its history. Judged by this standard, three extant races (or, rather, racial admixtures) are seen to be of peculiar worth: the Jewish, the Teutonic, and the Latin. All three of these, but especially the Jewish race, have shown by their history their high capacity for civilisation—and this whether they were pure, or, as was unquestionably the case as a rule, much mingled. Such as they are, they are tried and trusted." Fundamentally, however, this standard is neither self-evident nor unambiguous. If we propose to appraise the various races by the standard of their respective civilisations, we must first of all ask ourselves the significance and the value of the civilisation in question. For example, is western civilisation or Chinese civilisation the higher? As we shall show at much greater length in the sequel to the present volume, the present trend of western civilisation is towards the elimination of its creators and sustainers, and therefore towards self-destruction. We shall, perhaps, be told that only a "healthy" civilisation can be regarded as a standard of racial value. In that case the Chinese civilisation, which certainly tends much more than western civilisation to promote the survival of its sustainers, must be regarded as higher than western civilisation, and the race which has created it must be looked upon as higher than the Nordic race. Yet to draw such conclusions would be to argue in a circle. For, if we appraise the value of a civilisation by its effect upon the race, then we cannot proceed to appraise the value of the race by its effect upon the civilisation. The fact is that, though we ascribe to race as such a value of its own, no race can be regarded as either "higher" or "lower" than another, because all such estimates in a scale of altitude imply the application of some other standard of value than that of race per se. We cannot say that the earth stands higher or lower than the planet Mars, or that the earth is at the same level as Mars, because the concepts "high", "low", and "level", are coined with reference to the earth itself. So, too, is it with race. If we love our own race, not because of any particular civilisation, doctrine, or ethic, but simply for its own sake alone, this love makes it impossible for us to value any other race as greatly, and yet there is no implication that we therefore regard our own race as having a "higher" value in any objective sense.

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[1] Soziale Pathologie, third edition, Berlin, 1923, p. 481.

Of course the heredity factors of race are not the only causes of great cultural achievements. All civilisation, all achievement, is the outcome of a collaboration of racial endowments and environment. Racial biologists must guard against being as one-sided as are those who preach the supreme importance of environment. For a long time, now, practical persons have been concerned to know how the differences between human beings and the differences between civilisations have arisen. Rousseau believed that the decisive cause was to be found in differences in private property. This view has secured its clearest and most developed expression in what is known as the "materialist conception (or interpretation) of history" of Karl Marx and his followers, which still has numerous adherents to-day. In contraposition to this "materialist" doctrine there has, likewise since the eighteenth century, come into vogue the "idealist" or "spiritualist" conception or interpretation, according to which spiritual or mental achievements and ideals are the real motive forces of civilisation. But the champions of the "idealist" doctrine no less than the champions of the "materialist" doctrine overlook or positively deny that race is of any importance to civilisation. Although the "materialists" and the "idealists" constitute rival factions, engaged in fierce mutual conflict, they are in truth warring brethren, being children of one father, Lamarck. "Materialism" is substantially identical with that variety of Lamarckism which is known as "mechano-Lamarckism". According to this doctrine, all the differences between living beings, their adaptation and their evolution, are the outcome of the "direct influence" of environment. "Idealism", in its turn, in great measure coincides with "psycho-Lamarckism", a doctrine which ascribes the evolution of living creatures to mental forces, proclaiming that "the mind makes the body". The "materialists" tell us that the main differences among human beings consist in the differences between the possessing and the non-possessing; whilst the "idealists" tell us that the main differences consist in the differences between the educated and the uneducated. But the champions of these conflicting views regard the differences between human beings as bridgeable and eradicable. The "materialists" (the Marxists) teach that as soon as private property has been abolished, and as soon as (consequently) the economic differences between human beings have disappeared, all persons will become good and noble; the "idealists" dream of a general ennoblement of the human race by the inward appropriation of spiritual ideals, and in especial, of the idealist doctrine. Biologically, these two doctrines are equally untenable. The inequalities among human beings are mainly dependent upon the hereditary equipment, and this cannot be transformed in any simple way either by material or by spiritual influences. In the individual it cannot be changed at all, and in the race it can only be changed by selection. That contention, which shatters illusions, is regarded by the warring brethren as pessimistic, and they therefore join forces and make common cause against the biologists. The "idealists" declare that the biological view is "materialistic", which it is not; and the "materialists" often stigmatise it as high-flown idealism, which equally it is not. The modern biological outlook is fundamentally new, is something to which the old classifications and catchwords are inapplicable; and in its essential nature it is not pessimistic, for it, and it alone, points the way towards the sanation and stable advance of mankind and human civilisation.

The individualist conception of civilisation is only a variety of the idealist. Inasmuch as the individualists place great individuals in the foreground, they recognise, indeed, that civilisation has not been simply created by the masses, and also that it has not been created by economic conditions alone; but they usually overlook the fact that the creative energy of great individuals is the expression of their hereditary equipment, is the outcome of race.

An organic conception of civilisation must, indeed, give due weight to the conditions which are so one-sidedly emphasised by the "materialists", on the one hand, and the "idealists", on the other; but it must, as against materialists and idealists alike, insist that race, or in more general terms the hereditary equipment, is the first and indispensable condition of all civilisation. The Nordic thinker Kant, to whom the modern "idealists" especially appeal, was himself careful to avoid over-estimating the importance of environmental influences, insisting that "the inborn character is formed by the mingling of the blood in man, and the acquired and artificial character is only the outcome thereof".[1] The recognition that race is the substratum of all civilisation must not, however, lead any one to feel that membership of a superior race is a sort of comfortable couch on which he can go to sleep. For that reason I must not conclude my account of the mental peculiarities of the races without expressly insisting that the biological heritage of the mind is no more imperishable than the biological heritage of the body. If we continue to squander that biological mental heritage as we have been squandering it during the last few decades, it will not be many generations before we cease to be the superiors of the Mongols. Our ethnological studies must lead us, not to arrogance, but to action —to eugenics.

[1] Anthropologie in pragmatischer Hinsicht, Königsberg, 1798.

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The present work makes no claim to be exhaustive, so we renounce any attempt to give a complete bibliography, which would bulk far too largely. It will be enough to append a brief list of the most important and most comprehensive treatises bearing on the various sections of our book. For the use of readers who are not advanced students of the subject, critical notes are added regarding most of the publications named.

SECTION ONE

Baur, E. Einführung in die experimentelle Vererbungslehre, fifth and sixth editions, Berlin, 1922. [This is by the author of the first section of the present volume, and treats the same subject in fuller detail.]

Johannsen, W. Elemente der exakten Erblichkeitslehre, third edition, Jena, 1926. [Especially valuable as an introduction to the statistics of biological variation. We must demur, however, to the author's views concerning the significance of selection.]

Goldschmidt, R. Einführung in die Vererbungswissenschaft, fourth edition, Leipzig, 1923. [Extremely comprehensive, but we differ in some respects from the author. Bibliographies are appended to the chapters.]

Plate, L. Vererbungslehre, Leipzig, 1913. [In certain matters the author's opinions differ widely from those which we regard as sound. Contains a bibliography.]

Haecker, V. Allgemeine Vererbungslehre, third edition, Brunswick, 1921. [The relations between heredity and cytology are treated at great length. A bibliography is appended to each chapter.]

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Morgan, T. H. The Theory of the Gene, second edition, revised and enlarged, Yale University Press, 1928.

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Iltis, H. Gregor Johann Mendel, Leben, Werk und Wirkung, Springer, Berlin, 1924. [Part I contains a good account of Mendel's life and work; Part II, a presentation of Mendelism, includes an attempt at the impossible synthesis of Mendelism and Lamarckism (see above, p. 675).]

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"Zeitschrift für induktive Abstammungs- und Vererbungslehre", edited by E. Baur, and published by Börnträger, Berlin. [Deals chiefly with plants and the lower animals.]

"Archiv für Rassen- und Gesellschaftsbiologie", edited by A. Ploetz, and published by Lehmann, Munich. [Deals chiefly with man.]

"Annals of Eugenics", edited by Karl Pearson and E. M. Elderton, and published by the Cambridge University Press.

"Journal of Genetics", edited by W. Bateson and R. C. Punnett, and published by the Cambridge University Press.

"Genetics", issued by G. H. Schull, Brooklyn Botanical Gardens.

"Journal of Heredity", issued by the American Genetic Association, Washington.

"Hereditas", issued by R. Larsson, Lund. [Though this periodical is Scandinavian, most of the contributions are in German or English.]

"Genetica", edited by J. P. Lotsy, and published by Nijhoff, The Hague.

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Bauer, J. Vorlesungen über allgemeine Konstitutions- und Vererbungslehre, second edition, Berlin, 1923. [We can also recommend this work as an introduction to the subject, although we differ from the author about certain details relating to constitution and race.]

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